CLINICAL STUDY OF PRIMARY OPEN ANGLE GLAUCOMA IN DIABETIC PATIENTS

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CERTIFICATE

This is to certify that this dissertation entitled **"A CLINICAL STUDY OF PRIMARY OPEN ANGLE GLAUCOMA IN DIABETIC PATIENTS.**" has been done by ***DR.*** **R.VENKAT RAHAVAN** under my guidance in the Department of Ophthalmology, Madurai Medical College, Madurai.

I certify regarding the authenticity of the work done to prepare this dissertation.

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This is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfillment of the requirement for the award of M.S.,(Ophthalmology) Branch - III degree Examination to be held in APRIL 2012.

Place : Madurai
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LIST OF ABBREVIATIONS USED

ALT → Argon Laser Trabeculoplasty

BP → Blood Pressure

CT → Computerized Tomography

DM → Diabetes Mellitus

DR → Diabetic Retinopathy

HRT → Heidelberg Retinal Tomography

IOP → Intraocular pressure

MRI → Magnetic resonance imaging

NTG → Normal tension glaucoma

OH → Ocular Hypertension

OAG → Open Angle Glaucoma

POAG → Primary Open Angle Glaucoma

RBCs → Red Blood Corpuscles

RGC → Retinal Ganglion Cell

CLINICAL STUDY OF PRIMARY OPEN ANGLE GLAUCOMA IN DIABETIC PATIENTS

ABSTRACT

BACKGROUND

Primary open angle glaucoma is the commonest form of glaucoma accounting for atleast half of all the glaucomas.

It is also known as chronic open angle glaucoma and chronic simple glaucoma.

It is typically asymptomatic until significant visual field loss has occurred. Patients usually present with significant visual field loss in one eye and advanced disease in the other.

Diabetes Mellitus is one of the risk factors for POAG.

OBJECTIVES

• To study the hospital based prevalence of POAG among the diabetic patients attending Govt. Rajaji Hospital, Madurai Medical College, Madurai.

• To screen all diabetics for glaucoma.

METHODS

One hundred diabetic patients, both insulin dependent and non insulin dependent, above forty years of age, attending Govt. Rajaji Hospital, who came directly to Department of Ophthalmology or who were referred here for evaluation,

between May 2010 and August 2011, were screened for the detection of Primary Open Angle Glaucoma.

RESULTS

The results of the study show a clear evidence of an excess of POAG in diabetic population, which was 4..5 % .The prevalence among males was slightly more (5.1 %) as compared to females (3.12%).

In my study, the mean age of POAG among males was 56 yrs and 50.0 yrs among females. My studies show that the prevalence of POAG and the duration of DM was proportional and that the mean blood glucose level was higher in diabetics with POAG.

KEYWORDS

Primary open angle glaucoma; Diabetes Mellitus; Prevalence.

INTRODUCTION

The suggestion of an association between diabetes and Primary Open Angle Glaucoma (POAG) is not new. In 1971, Becker stated "Diabetes Mellitus occurs more often in patients with primary open angle glaucoma than in non-glaucomatous populations. Similarly, Glaucoma is more prevalent in diabetic than in non-diabetic population".

Diabetes Mellitus has been suggested as one of the risk factors of POAG along with other risk factors. Armstrong et al have reported a prevalence of POAG of 4.1% in the diabetic patients. The prevalence of diabetes in POAG was 1.7%. Many studies have shown a higher prevalence of elevated mean IOP and POAG among persons with diabetes compared to those without, and a higher prevalence of individuals with abnormal glucose metabolism among glaucoma patients than among the general population. Case-control studies have supported an association between diabetes and POAG.

It is tempting to accept diabetes as a definite risk factor for chronic open angle glaucoma, since diabetes is a disease of microangiopathy, and compromise of microcirculation of the optic disc is a possible contributing mechanism in the pathogenesis of glaucoma.

However, a number of studies including population-based investigations have not found an association between diabetes and open angle glaucoma. Population based prevalence data on association of glaucoma and diabetes among Asians are limited in number and of variable quality. Hence, my aim is to study the relation between the two in our region.

OBJECTIVES OF THE STUDY

• To study the hospital based prevalence of POAG among the diabetic patients attending GOVERNMENT RAJAJI HOSPITAL, Madurai.

• To screen all diabetics for glaucoma.

REVIEW OF LITERATURE

HISTORY OF GLAUCOMA

In the Hippocratic Aphorisms the term glaucoma was used to describe blindness coming on in advanced years associated with a glazed appearance of the pupil- “If the pupil becomes sea colored, sight is destroyed and blindness of the other eye often follows”.

Originally, indeed it was undifferentiated from cataract; both diseases were located in the lens, then considered the essential organ of vision, and both depended on a disturbance of the visual spirits.

The first suggestion of a disease associated with a rise in intraocular pressure and thus corresponding to what is, now known as glaucoma seems to occur in the Arabian writings of At-Tabari (tenth century) who wrote in the “Book of Hippocratic treatment” of a chronic inflammatory condition of the eye with raised tension, and of Sams-ad-Din (1348) of Cairo, who, among the one hundred and fifty three diseases of the eye and its adnexa, described with the ophthalmias a “migraine of the eye” or “headache of the pupil”, an illness associated with pain in the eye, hemicrania and dullness of the humours, and followed by dilatation of the pupil and cataract, if it became chronic,

tenseness of the eye and blindness supervened.

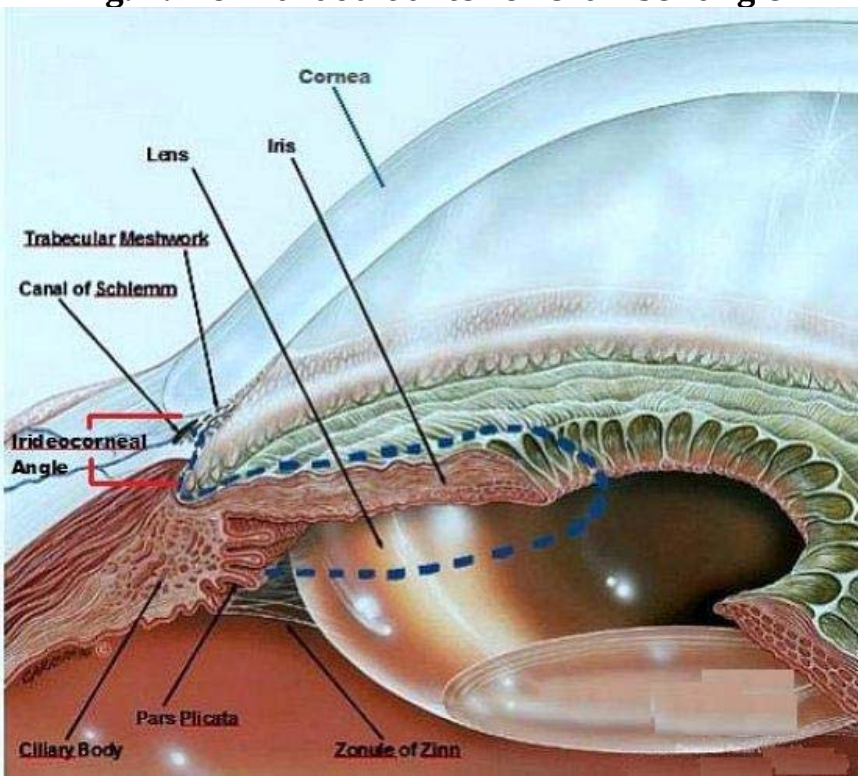
It was not until the beginning of the nineteenth century that the first excellent description of glaucoma with a raised ocular tension was given by Antoinne-Pierre Demours (1818) in a treatise, which incorporated the teaching of his father, Pierre Demours, and himself. The clinical picture was fully detailed and he described for the first time the appearance of the colors of a rainbow around the lights.

In London, Guthrie GJ recognized hardness of the eye as a characteristic of a disease, which he termed 'Glaucoma'. The next epoch in the history of glaucoma followed the introduction of the Ophthalmoscope, when clinical observation on the glaucomatous cup began to accumulate. The disease was divided into three categories - acute, chronic and secondary by Von Graefe (1857).

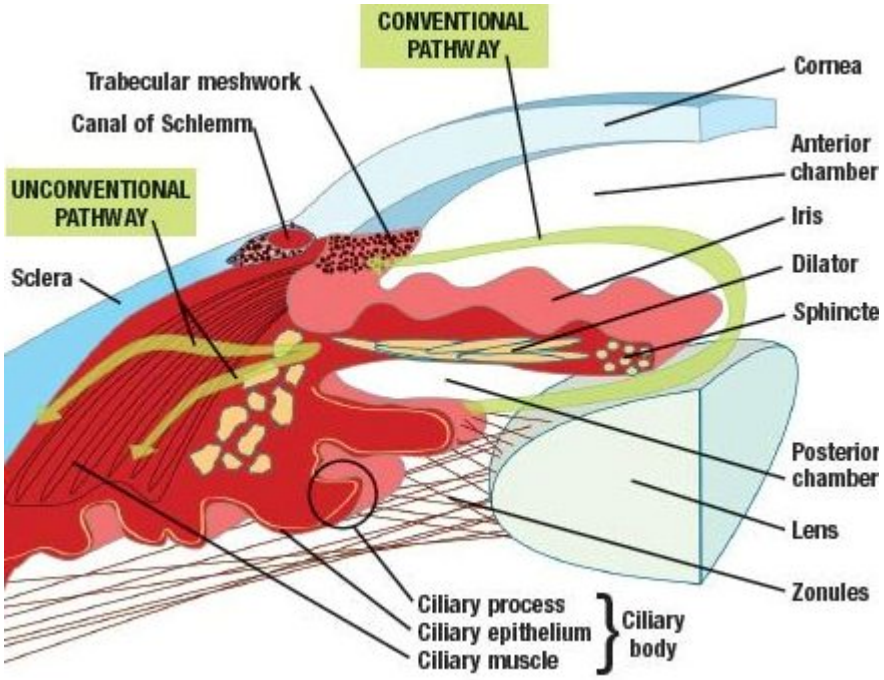
With the advent of the gonioscope, various others pointed out that glaucoma can be divided into two types, one with an open angle and other with closed angle. It was Otto Barkan (1938) of San Francisco, who suggested that angle closure glaucoma was due to obstruction of flow of aqueous through the pupil and advised a peripheral iridectomy to re-establish communication between the posterior chamber and anterior chamber as a means of surgical cure. From these revolutionary ideas grew the contemporary classification of glaucoma.

AQUEOUS HUMOUR

Fig. 1: Normal adult anterior chamber angle



Circulation and Drainage of Aqueous Humour



CLASSIFICATION OF GLAUCOMAS

Glaucomas can be classified based on:

1. The etiology

• i.e., the underlying disorder that leads to an alteration in aqueous humour dynamics.

2. The mechanism

• i.e, the specific alteration in the anterior chamber angle that leads to a rise in intraocular pressure.

Primary glaucomas

These are not consistently associated with obvious systemic or other ocular disorders that might account for this alteration.

• They are usually bilateral.

• They have a genetic basis.

Secondary glaucomas

These are associated with ocular or systemic abnormalities that appear to be responsible for the alteration in aqueous humour dynamics.

• They are unilateral or bilateral

• They can be inherited or acquired

CLASSIFICATION OF GLAUCOMAS BASED ON ETIOLOGY

A. PRIMARY GLAUCOMAS

i. Primary Open Angle Glaucoma

ii. Primary Angle Closure Glaucoma

B. DEVELOPMENTAL GLAUCOMAS

i. Primary Congenital Glaucoma

ii. Developmental glaucomas with associated anomalies

• Axenfeld-Rieger Syndrome

• Peter's Anomaly

• Aniridia

• Other developmental anomalies

C. SECONDARY GLAUCOMAS

i. Glaucomas associated with primary disorders of the corneal endothelium

• Iridocorneal endothelial syndrome

• Posterior polymorphous dystrophy

• Fuch's endothelial dystrophy

ii. Glaucomas associated with disorders of iris and ciliary body

• Pigmentary glaucoma • Iridoschisis • Plateau iris • Iris and ciliary body cysts

iii. Glaucomas associated with disorders of lens

• Exfoliation Syndrome

• Glaucomas associated with cataracts

• Glaucomas associated with lens dislocation

iv. Glaucomas associated with disorders of retina, vitreous, choroid

• Neovascular Glaucoma

• Glaucomas associated with retinal detachment and vitreoretinal abnormalities

v. Glaucomas associated with elevated episcleral venous pressure

vi. Glaucomas associated with intraocular tumours

• Malignant Melanoma • Retinoblastoma • Metastatic Carcinoma • Leukemias and lymphomas • Benign tumours

vii. Glaucomas associated with ocular inflammation

• Uveitis • Keratitis, episcleritis and scleritis

viii. Steroid – induced glaucomas

ix. Glaucomas associated with intraocular hemorrhage

x. Glaucomas associated with ocular trauma

xi. Glaucomas following ocular surgery

• Ciliary block (malignant) glaucoma

• Glaucomas in aphakia and pseudophakia

• Epithelial, fibrous and endothelial proliferation

• Glaucomas associated with corneal surgery

• Glaucomas associated with vitreoretinal surgery

CLASSIFICATION OF GLAUCOMAS BASED ON MECHANISM

A. OPEN ANGLE GLAUCOMAS

1. Primary Open Angle Glaucomas

2. Secondary Open Angle Glaucomas

a. *Pretrabecular forms* (membrane overgrowth)

i) Fibrovascular membrane (neovascular glaucoma)

ii) Endothelial layer, often with Descemet's like membrane

• Iridocorneal endothelial syndrome

• Posterior polymorphous dystrophy

• Penetrating and non penetrating trauma

iii) Epithelial down growth

iv) Fibrous ingrowth

v) Inflammatory membrane

• Fuch's heterochromic iridocyclitis • Leutic interstitial keratitis

b. Trabecular forms

i) Idiopathic

• Chronic open angle glaucomas

• Steroid-induced glaucomas

ii) Clogging of the trabecular meshwork

• **Red blood cells**

- Hemorrhagic glaucoma
- Ghost cell glaucoma

• **Macrophages**

- Hemolytic glaucoma
- Phacolytic glaucoma
- Malanomalytic glaucoma

• **Neoplastic cells**

- Malignant tumours
- Neurofibromatosis
- Nevus of Ota
- Juvenile xanthogranuloma

• **Pigment particles**

- Pigmentary glaucoma
- Exfoliation Syndrome (glaucoma capsulare)
- Uveitis
- Malignant Melanoma

• **Protein**

- Uveitis
- Lens-induced glaucoma

• **Viscoelastic agents**

• **Alpha-chymotrypsin induced glaucoma**

• **Vitreous**

iii. Alterations in the trabecular meshwork

a. Edema

• Uveitis (Trabeculitis) • Scleritis and episcleritis • Alkali burns

b. Trauma (angle recession)

c. Intraocular foreign bodies (Hemosiderosis, Chalcosis)

C. *Post trabecular forms*

i. Obstruction of Schlemm's canal

• Collapse of canal • Clogging of canal (e.g. Sickled RBCs)

ii. Elevated episcleral venous pressure

• Carotid cavernous fistula
• Cavernous Sinus Thrombosis
• Retrobulbar tumour
• Thyrotropic exophthalmos
• Superior vena cava obstruction
• Mediastinal tumours
• Sturge-Weber Syndrome
• Familial episcleral venous pressure elevation

3. Developmental Open Angle Glaucomas

a. Primary Congenital Glaucoma
b. Developmental glaucomas with associated anomalies

B. ANGLE CLOSURE GLAUCOMAS

1. Primary Angle Closure Glaucomas
2. Secondary Angle Closure Glaucomas

a. Anterior forms ("Pulling" mechanism)

i) Contracture of membranes

• Neovascular glaucoma

• Iridocorneal endothelial syndrome

• Posterior polymorphous dystrophy

• Penetrating and non penetrating trauma

ii) Contracture of inflammatory precipitates

b. Posterior forms (“Pushing” mechanism)

i) With pupillary block

• Pupillary block glaucoma

• Lens induced mechanisms

 Intumescent lens Subluxation of lens Mobile lens Syndrome

• Posterior synechiae

• Iris-vitreous block in aphakia

• Pseudophakia

• Uveitis

ii) Without pupillary block

• Plateau Iris Syndrome

• Ciliary block (Malignant) glaucoma

• Following lens extraction (forward vitreous shift)

• Following scleral buckling

• Following pan retinal photocoagulation

• Central retinal vein occlusion

• Intraocular tumours

 Malignant Melanoma Retinoblastoma

• Cysts of the iris and ciliary body

• Retrolenticular tissue contracture

 Retinopathy of prematurity Persistent hyperplastic Primary vitreous

c. Developmental angle closure glaucomas

i. High insertion of anterior uvea

• Congenital (infantile) glaucoma

• Juvenile glaucoma

• Glaucomas associated with other developmental anomalies

ii. Incomplete development of trabecular meshwork/Schlemm's canal

• Axenfeld-Rieger syndrome

• Peter's anomaly

• Glaucomas associated with other developmental anomalies

iii. Iridocorneal adhesions

• Broad strands (Axenfeld-Rieger Syndrome)

• Fine strands which contract to close angle (Aniridia)**4**

PRIMARY OPEN ANGLE GLAUCOMA

Primary open angle glaucoma is the commonest form of glaucoma accounting for atleast half of all the glaucomas. It is also known as chronic open angle glaucoma and chronic simple glaucoma. Although the exact

etiology is not known, it is likely that genetic defects set of a series of events that lead to increased resistance to aqueous outflow and increased vulnerability of the optic nerve head to a particular intra ocular pressure level. Within this large group of glaucomas, the most common form is typically defined by the following three criteria:

a. An intraocular pressure (IOP) consistently above 21mm Hg in atleast one eye

b. An open, normal appearing anterior chamber angle with no apparent ocular or systemic abnormality that might account for the elevated IOP

c. Typical glaucomatous visual field and /or optic nerve head damage**5**

POAG includes several forms:

I. "Senile sclerotic glaucoma" which is seen in the elderly and involves relatively low IOPs.

II. "High tension glaucoma" which occurs in a younger population and is marked by high pressures.

III. A third variation maybe " Normal tension glaucoma": in which the IOP never exceeds the statistical norms.

EPIDEMIOLOGY

Prevalence

Among Caucasian population POAG is:

1. 7% in Baltimore (1985-1988)

2. 1% in Beaver Dam, Wisconsin (1987-1988)

1.9% in country Roscommon, Ireland (1988-1989) and 1.1% in Rotter Dam, The Netherlands (1991-1993).

The Baltimore study and population based studies performed in St. Lucia, West Indies (1986) and Barbados, West Indies (1988-89) indicated that blacks have a prevalence of open angle glaucoma three or four times higher than that of whites, and the disease manifests at an earlier age among blacks. Age specific data showed a marked increase in glaucoma prevalence with age. Population based prevalence data on glaucoma among Asians are limited in number and of variable quality. Asia comprises heterogeneous groups of people and generalizations about all Asians cannot be made from results obtained from specific regions.

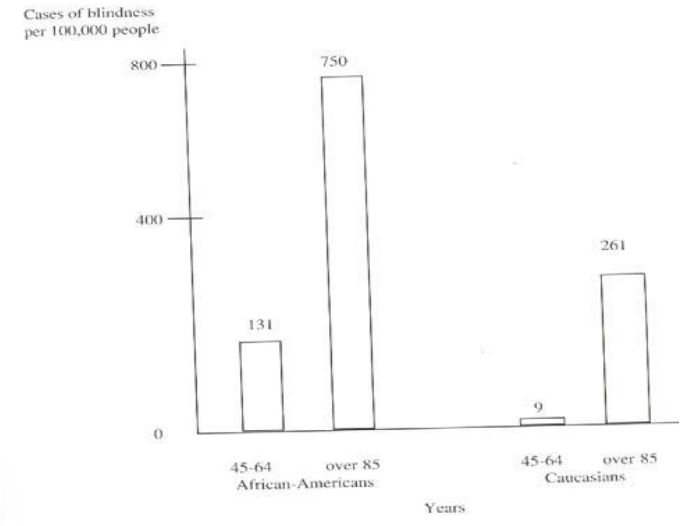
A population based survey conducted in Japan (1988-89) reported prevalence of 2.62% for open angle glaucoma. Among these, 78% had IOP below 21 mmHg. Their percentage is considerably higher than the 30 to 50% obtained in population based studies in the United States and Europe.

Incidence

There is a paucity of adequate data on incidence. Various studies have shown an incidence of 0.24% per year.

Blindness from Glaucoma

Fig. 3: Blindness caused by Glaucoma



The Model Reporting Area (MRA) study reported glaucoma blindness prevalence of 8.8 per 100,000 for whites and 131.4 per 100,000 for non-whites in the 45 to 64 age group. These estimates increased with age and were 261.4 per 100,000 for whites and 746.9 per 100,000 for non whites in the 85 or older age group.

Results from the Baltimore Eye Survey indicated that blindness due to open angle glaucoma increased with age among whites and blacks, began ten years earlier among blacks, and was the leading cause of non remediable blindness among blacks, with an overall age adjusted prevalence 6.6 times than among whites.

The overall incidence of new blindness considering the MRA data with crosssectional data from the Baltimore eye survey yields an estimate of 12,000 new cases of blindness every year from glaucoma in the United States.

Chronic open angle glaucoma as a health problem

Global health problem of primary open angle glaucoma as per world health organization estimations.

- People with high IOP (>21mm Hg) 104.65 million
- People with POAG 13.5million
- Incidence of Glaucoma per year 2.4 million
- Blindness prevalence for Glaucoma 5.2 million
- Blindness prevalence for POAG 3 million

The different types of glaucoma were theoretically calculated to be responsible for 15% of the blindness, placing glaucoma as the third leading cause of blindness worldwide.

RISK FACTORS

Risk factors with strong supporting evidence

Age

All studies agree that the prevalence of primary open angle glaucoma increases with the age of the population being considered. It is unusual for the

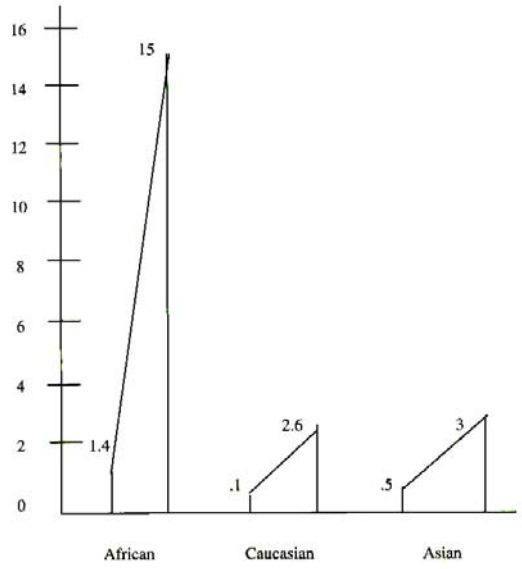
disorder to reach the clinical stage before the age of 40 (except in juvenile open angle glaucoma), and most cases are seen after age 65.

In one general population survey of 3,000 individuals, the prevalence of primary open angle glaucoma and low- tension glaucoma by age group was 0.22% among 40 to 49 year olds, 0.10% for 50 to 59, 0.57% or 60 to 69, 2.81% for 70 to 79 and 14.9% for patients above 80 years of age. Age, therefore becomes an increasingly significant risk factor with each decade.

The prevalence of POAG is 3-10 times higher among individuals older than 80 years (than people in their 40s).

Race

Fig 4: Prevalence of open angle glaucoma in different populations



Several studies have shown that primary open-angle glaucoma is more prevalent, develops at an earlier age, and is more severe in blacks as

compared to whites. Non-whites are said to have seven to eight times more blindness from glaucoma than whites. One possible explanation for this racial difference in susceptibility to elevated IOP might be the high incidence of sickle cell anemia among blacks, increasing the potential for optic nerve head ischemia. This theory, however was not supported in a

study that found sickle trait in only 2 of 40 blacks undergoing filtering surgery for primary open-angle glaucoma. It is hypothesized that larger cup disc ratios, large discs and more nerve fibres may be contributory. Asians, Canadians, Alaskans, Greenland Inuit Indians, and certain South American Indians are at an increased risk for narrow-angle glaucoma.

Sex

The prognostic significance of sex is less clear than that of age and race, although several studies suggest a higher prevalence among men. Women are at a greater risk for angle-closure glaucoma than men.

Family history

Heritable susceptibility has been shown. Between 10-20% of patients with glaucoma have a positive family history. Family history of glaucoma, especially in first-degree relatives is important. Family history of glaucoma in a sibling is the greatest risk factor, followed by glaucoma in a parent.

The Baltimore Eye Survey found that the relative risk of having glaucoma is increased 3.7- fold for individuals who have siblings with POAG. Genetics of glaucoma has been shown for the following:

• POAG – Juvenile onset; genetic linkage mapped to band 1q23.

• POAG – Adult onset

• Pigmentary glaucoma- Gene responsible for fragment dispersion syndrome mapped to band 7q35q36.

• Pseudoexfoliation

• Aniridia

• Axenfeld-Rieger Syndrome

• Primary congenital glaucoma**10**

Intraocular pressure

Intraocular pressure (IOP) might be defined as that pressure which does not lead to glaucomatous damage of the optic nerve head. IOP is a definite and important risk factor for developing glaucomatous damage but is not sufficient for a diagnosis. The prevalence of POAG is higher with increasing IOP. One tenth of patients with ocular hypertension develop field loss within 10 years. Each year, about 1% of all individuals with increased IOP progress to glaucomatous damage. As many as 50% of patients with glaucomatous optic neuropathy or visual field changes have IOP of less than 21 mmHg on

initial evaluation. Some eyes undergo damage at IOP of less than 18 mm Hg others tolerate IOP of more than 30 mmHg.

A pressure of 10-21 mmHg is considered normal; a non-gaussian distribution occurs with a skew towards higher pressures. 2-6 mmHg diurnal variation of IOP is normal. Greater than 10 mmHg variation is suggestive of glaucoma. Peak usually occurs in the morning hours.

Although the level of intraocular pressure (IOP) is one of the most consistent risk factors for the presence of glaucoma, the concept that statistically raised IOP is a defining characteristic for glaucoma has been almost universally discarded. This is based on several population based studies that document the typical disc and field damage of glaucoma in people with a statistically normal IOP and, conversely, people with statistically elevated IOP and no evidence of optic neuropathy.

The distribution of IOP in the general population as studied by Ley Decker (normal IOP was statistically defined two standard deviations above and below mean, as 11-21 mmHg) is not Gaussian, but is slightly skewed towards higher IOP's. Nearly 10% of the population over 40 years can have IOP's higher than 21 mm Hg, in the presence of open angles and normal optic discs. Such individuals, who were often labelled ocular hypertension, may not develop glaucoma.

In the 1996 edition of the American Academy of Ophthalmology, preferred practice pattern for POAG, elevation of IOP is neither a component of the definition of POAG nor a clinical characteristic of it. IOP has been reclassified as a risk factor for the clinical process rather than a clinical feature of POAG.

Risk factors with fair supporting evidence

Diabetes

Open-angle glaucoma (OAG) is an optic neuropathy characterized by progressive retinal ganglion cell (RGC) death and optic disc excavation. Evidence is accumulating that RGC apoptosis is the fundamental pathology of OAG. Among several risk factors for development and progression of OAG, inclusion of co-morbid diabetes has been controversial. Recent basic studies have shown that diabetes not only affects vascular tissues but also compromises neuronal and glial functions and metabolism in the retina, which ultimately gives rise to apoptotic death of retinal neurons including RGCs. The impaired metabolism of neurons and glia by diabetes may render RGCs susceptible to additional stresses related to OAG such as elevated intraocular pressure. Many studies including the Rotterdam study, Netherlands and the Blue Mountains eye study, Australia have investigated the possible existence of an association between glaucoma and diabetes.

The prevalence of POAG and ocular hypertension is several times higher in the diabetic population than in the general population. The prevalence of diabetes or a positive glucose tolerance test has also been shown to be higher in patients with POAG or a high IOP response to topical steroids. Diabetes also appears to influence the nature of visual field loss in patients with POAG, with a prevalence of inferior field loss of 64.4% versus 36.4% in diabetics versus non-diabetics, respectively, and a 32% prevalence of diabetes among POAG patients with primarily inferior loss, compared to 13% in those without such a defect.

Risk factors with weak supporting evidence

1. **Systemic hypertension**
2. **Migraine and Vasospasm**

PATHOGENESIS

Elevated intraocular pressure is a feature of POAG. A sustained increase in IOP may be due to increased formation of the aqueous humour, difficulty in its exit, or a raised pressure in the episcleral veins. Of these, the first and last rarely occur, and it follows that raised intraocular pressure is essentially due to an increased resistance to the circulation of the aqueous at the pupil and/or to its drainage through the angle of the anterior chamber. If the outflow pathway via the trabecular meshwork is blocked, some drainage

does occur through the uveoscleral outflow, but these alternative channels are not efficient and they are incapable of dealing with sudden changes of intraocular pressure.

Increased IOP in most cases is caused by decreased facility of aqueous humour outflow. Increased resistance or reduced facility of outflow is seen between anterior chamber and lumen of Schlemm's canal. Because trabecular meshwork prolapses into Schlemms' canal, it occludes the lumen and prevents circumferential flow of aqueous humour to collector channels. Obstruction of intrascleral collector channels by accumulation of glycosaminoglycans in adjacent sclera. If the trabecular meshwork or endothelium of Schlemm's is the site of increased resistance to outflow in POAG, then what process interferes with normal aqueous elimination?

Theories

a) Obstruction of trabecular meshwork by foreign material such as pigments, RBC's, glycosaminoglycans, amorphous material, extracellular lysosomes, plaque-like material and proteins.

b) Loss of trabecular endothelial cells.

c) Loss of giant vacuoles in inner wall of endothelium of Schlemm's canal.

d) Decreased permeability of trabecular meshwork

e) Loss of normal phagocytic activity, that is, the self – clearing filter property of the meshwork..

Increased resistance to outflow is linked with:

i) Altered corticosteroid metabolism Patients with POAG have

- Increased plasma levels of cortisol
- Increased suppression of plasma cortisol with different doses of exogeneous dexamethasone.
- Continued suppression of plasma cortisol by dexamethasone – despite concomitant administration of phenytoi.
- Disturbed pituitary adrenal function
- Increased inhibition of mitogen stimulated lymphocyte transformation by glucocorticoids.

ii) Dysfunctional adrenergic control

Reduced outflow in POAG is due to increased sensitivity to adrenergic agonists.

iii) Abnormal immunologic processes

Reduced outflow is due to increased levels of gamma–globulin and plasma cells in trabecular meshwork of patients with POAG; and increased antinuclear antibodies.

iv) Oxidative damage

Trabecular meshwork contains glutathione, which protects endothelial cells from effects of hydrogen peroxide and other oxidants.

Glaucomatous damage to optic nerve in most cases is a result of increased IOP. Some nerves, however, can withstand high pressures for a remarkably long time, whereas others seem to develop pathologic cupping at normal or even low normal intraocular pressures. Thus the resistance of nerve head to pressures is a key factor that determines whether an individual will develop progressive damage.

Three local characteristics of nerve head play a role in such resistance:

- Diameter of scleral ring
- Strength of lamina cribrosa
- Integrity of vascular supply

The pathogenesis of glaucomatous damage is attributed to

i) Vasogenic theory of nerve damage

This theory implies that structural and functional defects occurring in optic nerve head with glaucoma are mainly due to ischemia.

• Increased IOP leads to reduced capillary blood flow due to

a) Mechanical compression of vessels at lamina cribrosa

b) Reduced flow in annulus of Zinn, which supplies nutrition to laminar and post laminar optic nerve head, i.e. increased IOP leads to reduced perfusion pressure of the optic nerve.

• Recently, Anderson put forth the hypothesis that inhibition of autoregulation of blood supply to optic nerve can cause increased susceptibility of disc to pressure – induced ischemia.

ii) Mechanical theory of nerve damage

The coats of the eye can withstand fairly high intraocular pressures except at the lamina cribrosa - the fenestrated region through which optic nerve fibres enter the eye. Here, the nerve fibres are supported by glial tissue and have to blend over the edge of the disc.

Increased IOP leads to mechanical pressure on lamina cribrosa, altering capillary blood flow and reduced axoplasmic flow in the initial stages. Later, significant backward displacement and compaction of the laminar plates narrows the openings through which the axons pass, directly damaging the nerve fibre bundles, leading to atrophy. A reduction in blood supply follows the tissue atrophy because the atrophied tissue no longer requires nutrition.

Dysfunctional axoplasmic transport, because of these mechanical or vascular changes, leads to fewer trophic factors reaching the ganglion cells.

This causes damage and eventually death of the ganglion cells, which triggers apoptosis of adjacent cells.

Immunologic Studies

Increased gamma globulin and plasma cells in the trabecular meshwork of eyes with COAG have been reported, and a high percentage of patients with this disease were found in one study to have positive antinuclear antibodies reactions. These reports suggested a possible immunologic mechanism in the pathogenesis of COAG.

CLINICAL FEATURES

Symptoms

Primary open angle glaucoma is usually described as an insidious, slowly progressive, bilateral condition. The adjective "insidious" is appropriate because most patients are asymptomatic until the late stages of the disease. The few exceptions to this rule include the occasional patient who notices a scotoma when performing a monocular visual task, or the young patient who has sudden, severe elevations in intraocular pressure that cause corneal edema, halo vision and discomfort. If patients are not diagnosed until they develop extensive glaucomatous damage, they may become symptomatic from loss of fixation in one or both eyes or from loss of peripheral vision, which interferes with activities such as driving. The early stages of POAG

usually develop slowly over months to years. As glaucoma advances, however, the pace accelerates.

Signs

1. Elevated intraocular pressure

Most patients with POAG have elevated intraocular pressures in the range of 22 to 40 mmHg. Some patients have much higher pressures, which occasionally reach levels of 60 or even 80 mm Hg. It is important to remember that intraocular pressure fluctuates throughout the day and that patients with glaucoma undergo wider fluctuations than do normal individuals. Although most people reach their highest intraocular pressures in the morning, others may reach their peaks in the afternoon or evening or follow no consistent pattern. Diurnal intraocular pressure measurements maybe useful in some situations, including diagnosing primary open angle glaucoma, explaining progressive damage despite apparent good pressure control, evaluating the efficacy of therapy, and distinguishing normal tension glaucoma from primary open angle glaucoma.

Most individuals have fairly symmetric intraocular pressure readings. When pressure is higher in one eye, that eye usually has a larger cup and a more damaged visual field than the fellow eye.

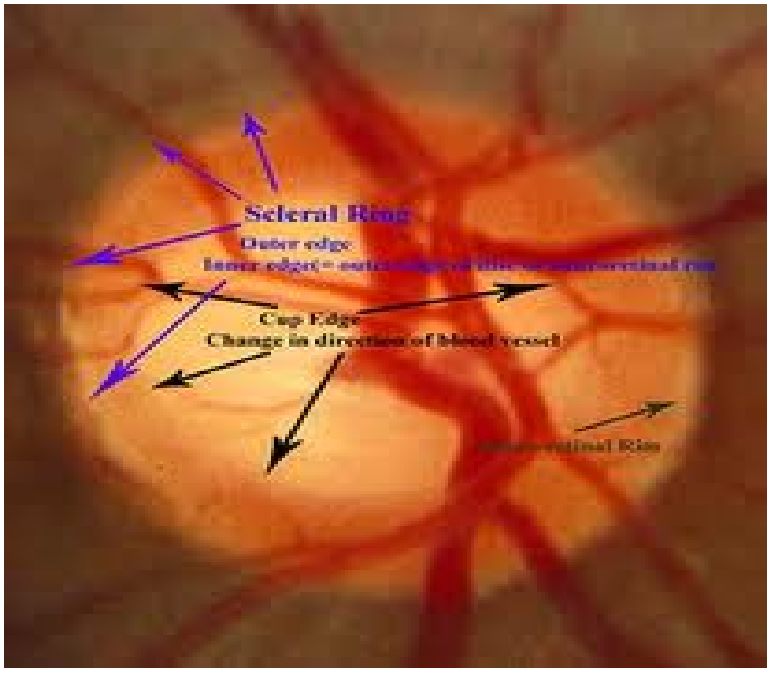


Fig. 1: Normal optic Disc

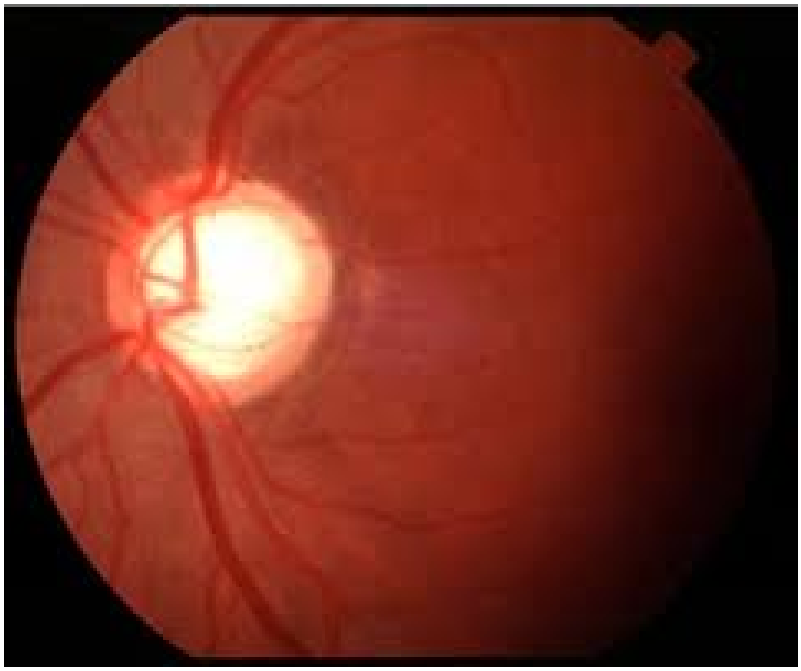


Fig. 2: Glaucomatous optic Disc

2. Optic disc changes

The most important variables for early detection of glaucomatous optic nerve damage in ocular hypertensive eyes before the development of visual field loss are:

a. Shape of neuroretinal rim

b. Size of optic cup in relation to size of the optic disc

c. Decreased visibility of the retinal nerve fibre layer

d. Occurrence of localized retinal nerve fibre layer defects and hemorrhages

As bundles of axons are destroyed in an eye with glaucoma, the neural rim begins to thin in one of several patterns.

i) Focal atrophy: The inferior temporal rim becomes thinner than the superior temporal rim. The vertical cup-disc ratio becomes more than the horizontal cup disc ratio. The changes in chronological order are:

Polar notching

The atrophy of the neural rim often begins as a small discrete defect usually in the inferior temporal quadrant. This is also known as focal notching or pit like change (pseudopit).

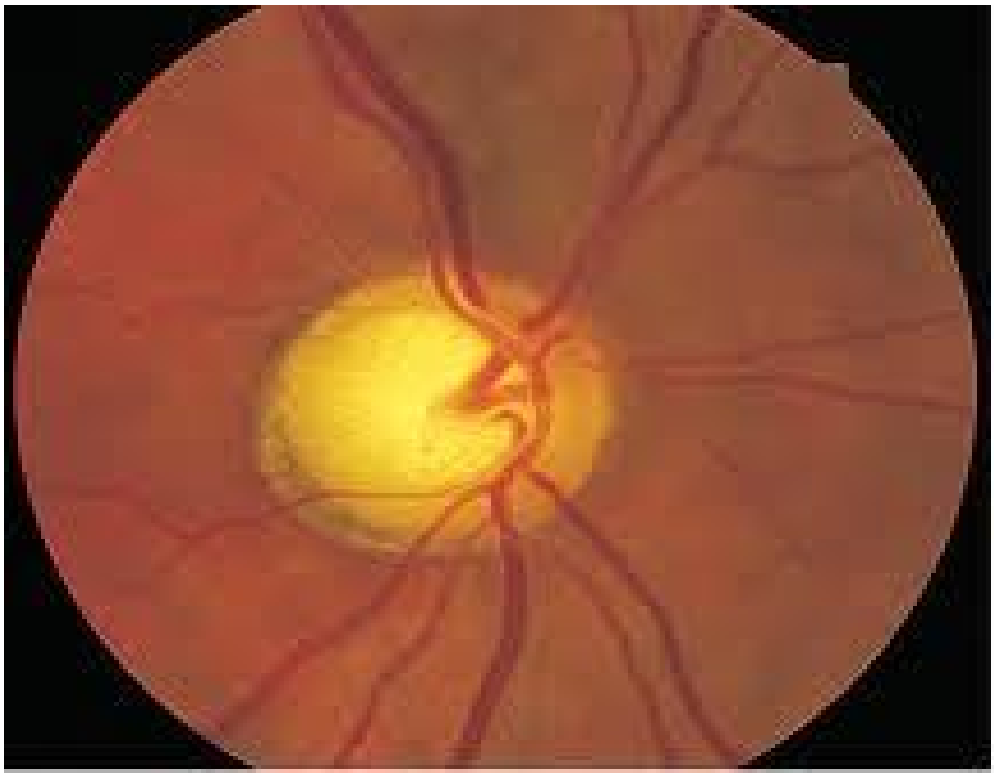


Fig.3: Focal Saucerization

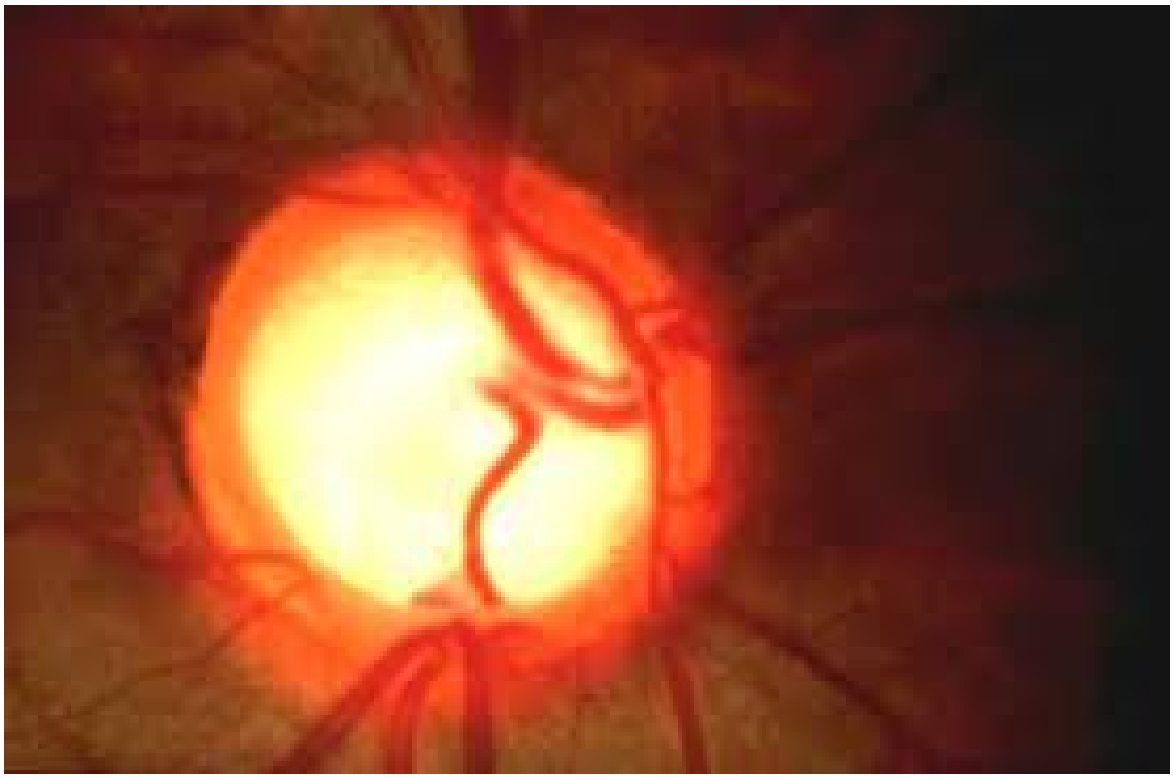


Fig. 4: Bayonetting sign

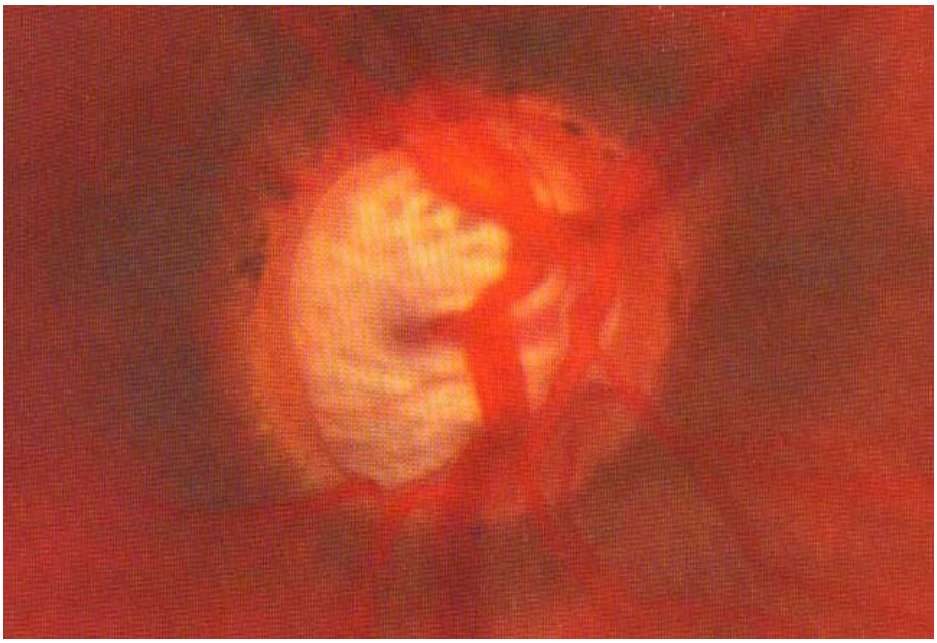


Fig.7 Laminar dot sign

Fig. 8: Focal notching

Sharpened polar neural edge:

As the focal notching enlarges and deepens, it may develop a sharp nasal margin often adjacent to a major vessel.

Sharpened rim:

When local thinning reaches the disc margin there will be no visible neural rim in that area.

Bayoneting sign:

If a retinal vessel crosses the sharpened rim, it will bend sharply at the edge of the disc.

ii) Concentric atrophy: Enlargement of the cup in concentric circles, most often directed inferotemporally or superotemporally.

Temporal unfolding: Loss of the neural rim tissue usually begins temporally and then progresses circumferentially towards the poles. This has been called temporal unfolding. This is very difficult to differentiate from the physiologic cup.

Cresentic shadow: A thinning of the neural rim maybe seen as a cresentic shadow adjacent to the disc margin as the intense beam of a direct ophthalmoscope passes across the neural rim. It should not be confused with the grey crescent.

iii) Deepening of the cup: In some cases the predominant pattern of early

glaucomatous atrophy is a deepening of the cup.

Overpass cupping: The vessels initially bridge the deepened cup and later collapse into it.

Laminar dot sign: Exposure of lamina cribrosa by the deepening cup is recognized by grey fenestra of lamina, which has been referred to as laminar dot sign.

iv) Pallor /Cup discrepancy

In the early stage of glaucomatous optic atrophy, enlargement of the cup may progress ahead of that of the area of pallor. This biphasic pattern differs from other causes of optic atrophy in which the area of pallor is typically larger than the cup.

Saucerization: Refers to a pattern of glaucomatous change in which diffuse, shallow cupping extends to the disc margin with retention of central pale cup.

Focal saucerization: More localized, shallow, sloping cup.

Tinted hallow: Retention of normal neural rim colour in the area of focal saucerization has been called tinted hallow.

Shadow sign:As the glaucomatous damage progresses, the colour is replaced by grayish hue termed shadow sign.

*v) Advanced glaucomatous cupping**:*** Bean-pot cupping: In advanced cases all the neural rim tissue is lost ;the result is total cupping, which is seen clinically

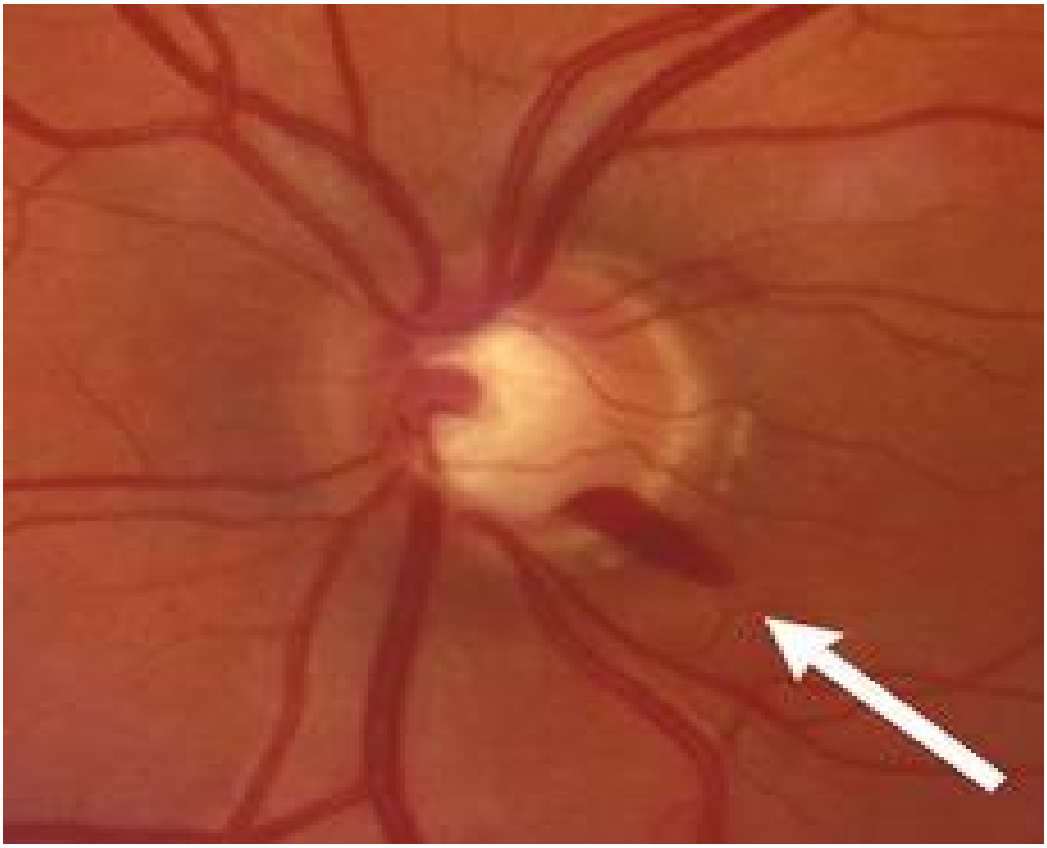


Fig.5 Drance Hemorrhage

Fig 6. Advanced cupping in concentric atrophy end stage glaucoma

as a white disc with loss of all neural rim tissue and bending of vessels at the margin of the disc. It is called bean pot because on histologic examination, there will be a posterior displacement of lamina cribrosa and undermining of the disc margin.

Vascular Signs

i) **Optic disc hemorrhages**

Splinter hemorrhages, usually near the margin of the optic nerve head are a common feature of glaucomatous damage..

ii) **Tortuosity of retinal vessels**

Maybe seen on the disc with advanced glaucomatous optic atrophy. It represents loops of collateral vessels in response to chronic central retinal vessel occlusion.

iii) **Location of retinal vessels in relation to the cup**

a) **Overpass cupping**: The vessels initially bridge the deepened cup and later collapse into it.

b) **Baring of the circumlinear vessels**: In many normal optic nerve heads one or two vessels may curve to outline a portion of the physiologic cup. With glaucomatous enlargement of the cup, the circumlinear vessels maybe ‘bared’ from the margin of the cup.

iv) **Nasal displacement of the retinal vessels**

Does not provide a useful diagnostic parameter.

Peripapillary changes associated with glaucomatous optic atrophy

i) **Nerve fibre bundle defects**

The loss of axonal bundles produces visible defects in nerve fibre layer which appears as dark stripes of varying width in the peripapillary area paralleling the normal retinal striations.

ii) **Peripapillary depigmentation**

Seen both in normal as well as patients with glaucoma. It is called as peripapillary halo, peripapillary atrophy or chorioscleral crescent. It occurs more frequently and is larger in glaucomatous damage as compared to normal eyes and it has been observed to enlarge progressively in eyes with glaucoma.

3. Gonioscopy

By traditional definition, the anterior chamber angle in eyes with POAG is open and grossly normal. Preliminary studies, however, suggest that these patients may have more iris processes, a higher insertion of the iris root, more trabecular meshwork pigmentation and a greater than normal degree of segmentation in the pigmentation of the meshwork.

4. Visual field abnormalities

a. Generalized depression of visual field

Diffuse reduction in visual threshold is one of the earliest detectable alterations in the visual field of a patient with glaucoma. But they are non-specific of glaucoma. Concentric contraction of the visual field, which is more marked in the nasal field, is called "crowding of the peripheral nasal isoptres".

Enlargement of the blind spot: also considered as an early field change which is non specific for glaucoma.

Angioscotoma: are long, branching scotomas above and below the blind spot, which are presumed to result from shadows created by large retinal vessels. Other measures of generalized visual impairment in glaucoma: Other psychological tests that support the concept that a generalized decrease in visual function may be the earliest visual change in glaucoma are

i. **Colour vision**

Reduced sensitivity to colour may precede any detectable loss of peripheral or central vision. The colour vision deficit is associated primarily with blue sensitive pathways. The clinical usefulness of colour vision in the early detection of glaucoma is limited by a lack of sensitivity and specificity.

ii. **Contrast sensitivity**

Subtle loss of both central and peripheral vision can be demonstrated in some patients with glaucoma prior to detectable visual field change.

iii. **Electrophysiological studies**

Electroretinography shows reduced amplitude.

Visually Evoked Potential – Reduced response to high flicker VEP

Electrooculography – shows increased baseline values

iv. **Miscellaneous tests**

Dark adaptation – is abnormal.

A relative afferent pupillary defect.

Reduced brightness comparison test.

b. ***Nerve fibre bundle defects***

Focal defects resulting in loss or impairment of retinal nerve fibre bundles are more specific for glaucoma and constitute the most definitive early evidence of visual field loss from these disorders.

i. **Scatter**: Areas where there is a variable threshold response to repeated testing in the same area. This has been referred as scatter, fluctuation or localized minor effects. Scatter is not a definite sign of glaucoma.

ii. **Arcuate defect**: Early visual loss in glaucoma commonly occurs within arcuate area. They most often appear first as one or more localized defects or paracentral scotoma.

• *Siedel's scotoma*: Early arcuate defect may connect with the blind spot and taper to a point in a slightly curved course.

• *Arcuate or Bjerrum scotoma*: As the isolated defects enlarge and coalesce, they form an arching scotoma that eventually fills the entire arcuate area from the blind spot to the median raphe.

• *Ring scotoma or double arcuate scotoma*: When two arcuate scotoma from above and below meet.

• *Nasal step*: The loss of retinal nerve fibres rarely proceeds at the same rate in the upper and lower portions of the eye. Consequently, a step like defect is frequently created where the nerve fibres meet along the median raphe. Since the superior field is involved more frequently than the inferior portion in the early stages of glaucoma, most common is the superior nasal step.

• *Central nasal step*: Created at the nasal termination of unequal double arcuate scotoma.

iii. **Peripheral field defects**

- Peripheral nasal step of Roenne: Unequal contraction of peripheral isoptres produce this defect.

- Vertical step: A stepwise defect along vertical midline is referred to as vertical step or hemianopic offset. It is less common.
- Temporal sector defect: The retinal nerve fibres nasal to the optic nerve head converges on the disc by a direct route. A lesion involving these fibre bundles produce a sector defect temporal to the blind spot. They usually appear in later stages of glaucoma.

c. ***Advanced glaucomatous field change***

Visual field is reduced to central and temporal island of vision. The temporal island of vision is more resistant and persists long after central vision is lost.

Correlation between optic nerve head and visual field defects

Clinically recognizable disc changes precede detectable field loss. The extent of axonal loss is much greater than the corresponding visual field change. Upto 35% of the fibres maybe gone in one eye with normal field, while more than half the fibres maybe lost by the time reproducible early field defects are found, and 10% of fewer axons may remain by the stage of severe field loss. The nature of optic nerve head cupping can also be used to predict the type of field loss. Extensive or focal absence of neural rim tissue especially at the inferior or superior pole is the most reliable indicator of

visual field disturbance and is usually associated with a field defect in the corresponding arcuate area.

In general, optic nerve head and retinal nerve fibre layer changes have their greatest value in the early stages of glaucoma, while progressive visual field loss becomes the more useful guide to therapy in advanced cases.

DIAGNOSIS

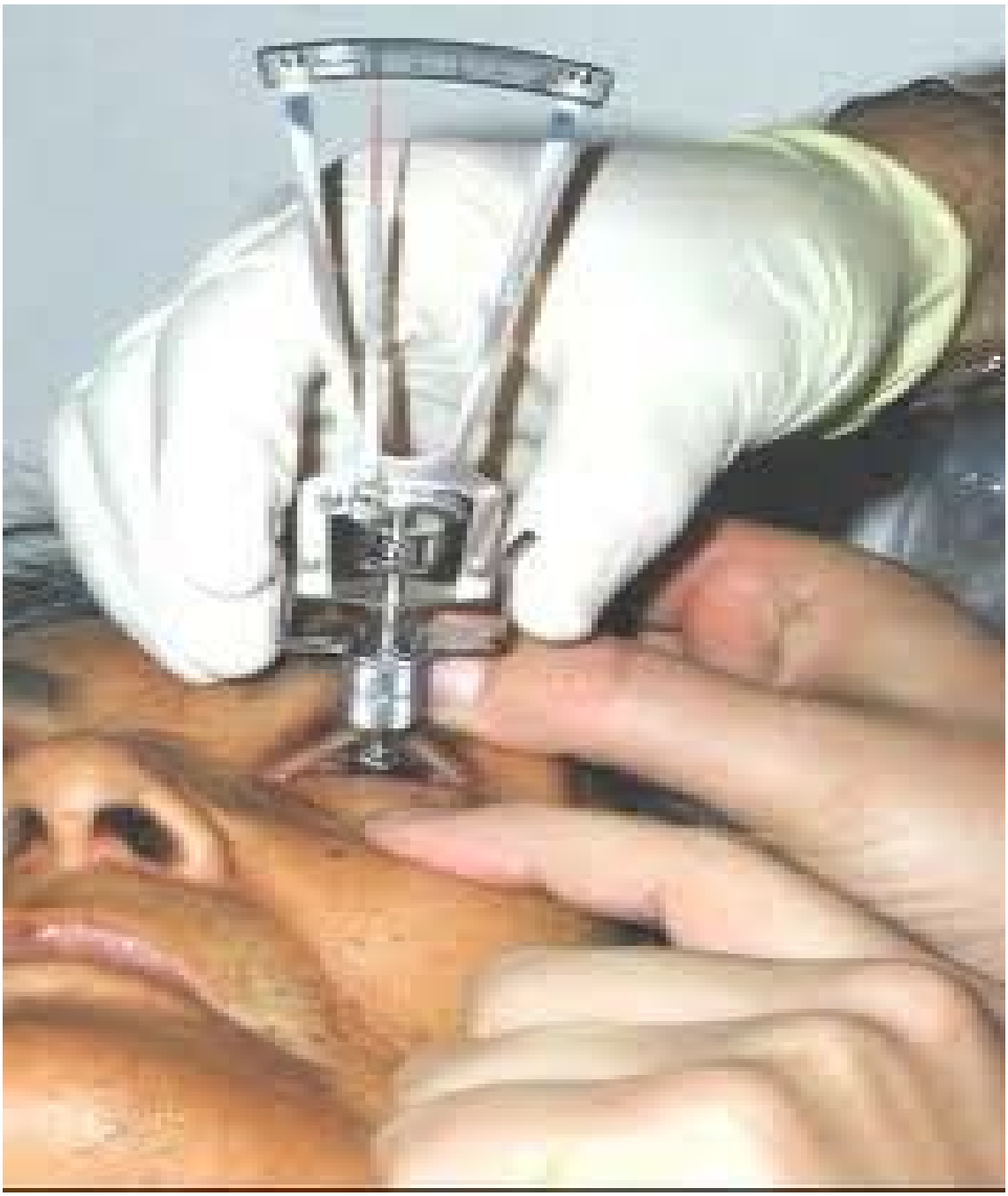
A diagnosis of POAG can be made after performing the following tests:

1. Intraocular pressure recording
2. Optic nerve head assay
3. Gonioscopy
4. Visual field analysis

Intraocular pressure recording

Tonometer is the instrument used to measure the intraocular pressure. All clinical tonometers measure the IOP by relating a deformation of the globe to the force responsible for the deformation. The two basic types of tonometers differ according to the shape of the deformation: indentation and applanation (flattening).

1. **Schiotz Indentation Tonometer - a known weight used to indent the cornea.**
2. **Applanation Tonometers**

3. 

4. Fig. 9 Schiotz Tonometry being performed

The shape of deformation with these tonometers is a simple flattening, and because the shape is constant, its relationship to the IOP can be derived from mathematical calculations.

Goldmann applanation Tonometry

Goldmann based his concept on tonometry on a modification of the Maklakov- Fick law (also referred to as the Imbert Fick Law). This law states that an external force (W) against a sphere equals the pressure in the sphere (P1) times the area flattened (applanated) by the external force (A): W=P1 x A It was necessary to modify the Imbert-Fick law in the following manner to account for the characteristics of the cornea: W+S=P1A1 + B

Where A1 =7.35 mm2, S balances B and W=P. The instrument is mounted on a standard slit lamp in such a way that the examiner's view is directed through the center of a plastic biprism, which is used to applanate the cornea. Two beam –splitting prisms within the applanating unit optically convert the circular area of corneal contact into semicircles. The prisms are adjusted so that the inner margins of the semicircles overlap when 3.06 mm of cornea is applanated. Other applanation tonometers with variable force:

1.Hand-held Goldmann-type tonometers

2.Perkins Applanation tonometer:

3.Drager Applanation tonometer:

4.Mackay Marg Tonometer:

5.Tono-Pen:

6.Pneumatic tonometers:

7.Non-contact Tonometer

Diurnal Variation

The IOP varies with age, time of the day and with the measurement methods. This variation in the IOP is very significant for the management of POAG. A wide fluctuation of IOP on diurnal measurement is suggestive of glaucoma and a control of the peak pressure is essential for a good control of POAG. These measurements of IOP should be conducted over 24 hours on an inpatient, outpatient or self-tonometry basis.

Provocative Tests

The water-drinking test is a test in which outflow channels of the eye are stressed to provoke a distinction between normal eyes and those with risk of POAG. Spaeth and Vacharat have shown that the rise of pressure in this test is dependent on the baseline IOP and so a positive test could have a correlation to glaucomatous field changes.

Optic Nerve Head Assay

Stereophotography of the optic nerve head is at present the best way to ascertain small changes sequentially. A diagrammatic representation of the

disc, detailing the cupdisc ratio, status of the neuroretinal rim, e.g. width, irregularity, colour, vascular alterations and a documentation of any nerve layer defects at every follow-up should be mandatory.

Other methods of assessing nerve fibre layer

In the clinical evaluation of the optic nerve head, the direct ophthalmoscope is very useful, especially when looking through a small pupil or evaluating the nerve fibre layer with a red-free filter (green-filter ophthalmoscopy).The other methods used are :

Stereochromoscopy

Stereochromoscopy utilizes the stereoscopic principle to detect subtle changes in photographs of a disc taken at different times. If there has been any progression of the cupping, the disparity in the cup margins of the superimposed photographs will produce a stereoscopic effect. A modification of this concept, referred to as stereochronometry, uses a stereo plotter to measure the changes created by the two photos. Other techniques for detecting differences in serial fundus photographs involve analysis of flicker while alternately viewing one photograph and then the other, and electronic subtraction, in which areas of disparity between two images will be enhanced.

Colorimetric Measurements

Colorimetric measurements have also been studied in an effort to detect reduced or changing colour intensity of the optic nerve head. A photographic technique has also been developed to permit quantitative evaluation of the relative brightness of the illuminated optic nerve head.

Ultrasonography

Ultrasound can be used to detect glaucomatous cupping of 0.7 cup/disc ratio or greater, and high –resolution contact B –scan echography may one day provide a reliable estimate of the optic cup size in eyes with opaque media.

Confocal Laser Scanning

This is a technique for obtaining high-resolution images by using a focused laser beam to scan over the area of the fundus to be imaged. By scanning the fundus with the laser, a two-dimensional image can be built up as an array of pixels (confocal scanning laser ophthalmoscopy). If a series of such images are obtained at successive planes of depth in the tissue, these can be used to construct a three-dimensional image (confocal scanning laser tomography).

Optical Retinal Tomography

The principle of optical coherence tomography (OCT) involves a low-coherence infrared (843 nm) diode light source, which is divided into

reference and sample paths. Reflected sample light from the subject's eye creates an interference signal with the reference beam, which is detected in a fibreoptic interferometer. Cross-sectional images of the retina and disc are then constructed from a sequence of signals, similar to an ultrasound B mode. Preliminary studies have demonstrated that nerve fibre layer thickness can be measured using the OCT.

Heidelberg Retinal Tomography

Conventional perimetry is insensitive to ganglion cell loss. 50% of ganglion cells are located 20 degrees around fovea. Heidelberg Retinal Tomography (HRT) is a confocal scanning laser ophthalmoscope for acquiring and analyzing three-dimensional images of posterior segment. The optic nerve head and peripapillary retina are scanned in two directions. In thirty-two planes, two million data points are obtained and all are reconstructed to give a topographic picture.

Rasterstereography

"Raster" simply refers to a scanning pattern that moves from side-to-side and from top-to-bottom (the same scanning pattern used in confocal laser scanning). In rasterstereography, a series of horizontal dark/light line pairs are projected on the disc and peripapillary retina at a fixed angle, and the computer scans a video image of the lines in a raster fashion. Since the lines

are deflected proportional to the height or depth of the disc and retinal surfaces, a computer algorithm can translate the deflections into depth numbers and create a topographic map.

Gonioscopy

It is an important confirmatory technique, distinguishing POAG from creeping angle closure glaucoma and other secondary glaucomas. This is performed using an indirect goniolens of either the Goldmann or Zeiss 4 mirror type.

Perimetry

The term perimetry is used to describe techniques employed to examine and quantify the visual field using targets of various sizes and colours. Visual field testing is generally performed by adapting the eye fully corrected for the testing distance with full field lenses, to a photoptic background illuminiscence over which is superimposed a brighter test stimulus in a kinetic or static manner.

This maybe accomplished by using kinetic and /or static techniques with instruments that are either manually operated or computer-assisted (automatic).

Kinetic techniques

This method involves moving the test object from a non-seeing to a seeing area and recording the point at which it is first seen in relation to fixation. The procedure documents the boundaries of the visual field, for both the absolute limits as well as area of relative differences in visual acuity within the field. Types of kinetic perimeters are Listers' perimeter and Goldmann's perimeter.

Static techniques

This approach involves the presentation of stationary test objects utilizing either suprathreshold or threshold presentations.

Goldmann perimetry is the present standard for normal visual field assessment, allowing standardization of background illumination and stimulus intensity, reproducible positioning of the stimulus and fixation monitoring. For Goldmann perimetry the patient should be comfortably positioned and the threshold determined on the 25 degrees isopter, 15 degrees above and below the temporal horizontal meridian. 12e is the standard test stimulus used for the central 5-15 degrees isopters and the nasal and temporal meridians, which are, examined minutely both by static and kinetic perimetry techniques. The depth of any scotoma, relative or absolute, is ascertained by gradually increasing stimulus size / intensity. The peripheral field is charted using the 14e

stimulus. Automated computerized perimetry is a more accurate and more informative technique for glaucomatous field evaluation.

Automated Perimeters

Automated computerized perimetry is a more accurate and informative technique for glaucomatous field evaluation. Those, which have received the most reported investigation, are the Delta program with the Octopus perimeter and the STATPAC with the Humphrey Field Analyzer. In case of the Humphrey Field Analyzer, the STATPAC uses a large normal database, while STATPAC II uses a database of stable glaucoma patients. The STATPAC II also includes linear regression analysis and glaucoma change probability. A third statistical algorithm with the Humphrey Field Analyzer is the Progressor Program for analysis of serial fields downloaded to a personal computer.

MANAGEMENT

Indications for treatment

Generally, the ophthalmologist institutes treatment when the patient has the classic glaucoma triad of visual field loss, optic nerve cupping and elevated intraocular pressure.

Goals of treatment

No definitive procedure exists for POAG, so one must aim at controlling the disease rather than curing it. When treating POAG, the

ophthalmologist attempts to stop the progression of the disease or to slow it sufficiently to maintain good vision for the patient's lifetime. The goal is usually achieved by lowering intraocular pressure.

Medical therapy

POAG has traditionally been thought of as a medical disease. The basic principle of medical therapy is to use the least amount of medicine that will control the glaucoma with the fewest side effects.

Anti-glaucoma drugs

Classification

• Parasympathomimetic drugs (Miotics)

• Sympathomimetic drugs (adrenergic agonists)

• Beta blockers

• Carbonic anhydrase inhibitors

• Hyperosmotic agents

• Prostaglandins

• Calcium channel blocker

Parasympathomimetic drugs

Preparations:

1. Pilocarpine: 1%, 2% and 4% eye drops 4% gel Ocuserts

2. Carbachol: 0.75% and 3% eye drops

3. Ecothiophate iodide: 0.03, 0.06 and 0,125% eye drops

4. Demecarium bromide: 0.125% ,0.25% eye drops

5. Physostigmine: 0.5% ointment

Sympathomimetic drugs

They act by stimulation of alpha, beta or both the receptors.

Preparations

1. Epinephrine: 0.5%, 1% and 2% eye drops

2. Dipivefrine: 0.1% eye drops

3. Clonidine hydrochloride: 0.125% and 0.25% eye drops

4. Brimonidine 0.2% eye drops

5. Apraclonidine : 0.5% and 1% drops

Beta-adrenergic blockers

These are presently the most frequently used anti-glaucoma drugs.

Preparations

1. Timolol: 0.25% and 0.5% eye drops

2. Betaxolol: Cardioselective beta-blockers

0.25% and 0.5% suspension

3. Levobunolol: 0.5% eye drops

4. Carteolol: 1% and 2% eye drops

5. Metipronolol: 0.1%, 0.3% and 0.6% eye drops

Carbonic anhydrase inhibitor (CAIs)

These are potent and most commonly used anti-glaucoma drugs.

Preparations:

1. Acetazolamide: 250 mg tablets

2. Dichlorphenamide: 50mg tablets

3. Methazolamide: 50mg tablets

4. Ethoxzolamide: 125 mg tablets

5. Dorzolamide: Topical carbonic anhydrase inhibitor 2% eye drops

Hyperosmotic agents

These are second class of compounds, which are administered systemically to lower the IOP.

Preparations:

1. Glycerol: 50% solution
2. Mannitol: 20% solution
3. Urea
4. Isosorbide

Prostaglandin derivatives

Latanoprost : 0.005%

Mechanism of action:

1. Increasing uveoscleral outflow

2. Causes reduction in episcleral venous pressure

Calcium channel blockers

Verapamil 0.125% and 0.25% eye drops Mechanism of action: Not known. It might be due to its effects on secretory ciliary epithelium.

Tolerance and compliance with medical therapy

Surgical intervention is usually indicated whenever there is progressive glaucomatous damage despite "maximum tolerable medical therapy".

Surgical intervention

When the glaucoma is uncontrolled medically, laser trabeculoplasty is usually the first surgical procedure of choice, followed by incisional surgical intervention when necessary. Trabeculectomy is usually the preferred incisional surgical technique for POAG.

Trabeculectomy lowers the intraocular pressure by creating a fistula between the inner compartments of the eye and the subconjunctival space (i.e., filtering bleb). The fistula is protected or guarded by a superficial scleral flap.

In non-penetrating filtration surgery, the anterior chamber is not entered, thusreducing the incidence of postoperative over filtration and hypotony. The two currently used procedures are *deep sclerostomy,* in which bleb formation often occurs and *viscocanalostomy,* in which bleb formation is infrequent. Aqueous drainage devices are generally reserved as a last resort

for patients with glaucoma that is refractory to standard filtering surgery. This includes patients with extensive conjunctival scarring, chronic inflammation and ocular trauma. IOP lowering with glaucoma drainage devices is generally not as effective as with filtering surgery. Cyclophotocoagulation is another alternative for patients with glaucoma that is refractory to other interventions and where the visual potential is poor.

METHODOLOGY

Two hundred diabetic patients, both insulin dependent and non insulin dependent, above forty years of age, attending GOVT. RAJAJI HOSPITAL Madurai, who came directly to Department of Ophthalmology or who were referred here for evaluation, between May 2010 and August 2011, were screened for the detection of Primary Open Angle Glaucoma.

Patients who satisfied any one of the inclusion criteria were selected.

Inclusion criteria

• IOP > 21 mmHg (by Schiotz tonometry) with visual field defects.

• IOP > 21 mmHg (by Schiotz tonometry) with optic nerve head changes.

• Optic nerve head changes with visual field defects.

• Normal IOP with no visual field defects or optic nerve head changes, with asymmetry of IOP in both eyes of > 5 mmHg.

Exclusion criteria

• Closed angle on gonioscopy

• Drug induced (corticosteroids)

The diabetic patients above 40 years of age were briefly explained about the study and the tests they would have to undergo. These patients were subjected to detailed eye examination in the Department of Ophthalmology, Govt. Rajaji Hospital Madurai.

These examinations include:

- Visual acuity
- Slit lamp examination
- Tonometry with Schiotz tonometer, diurnal testing
- Ophthalmoscopy
- Gonioscopy
- Visual field testing using Automated perimeter (Octopus /Humphrey)

Patients with significant disc cupping (and other signs of glaucomatous disc changes), with field defects, regardless of IOP were suspected as having POAG. IOP was recorded to allow distinction between POAG with elevated pressure and Normal Tension Glaucoma. Criteria for diagnosis of ocular hypertension were pressure greater than 21 mmHg, with no disc changes, and in the absence of field defect.

METHOD OF STATISTICAL ANALYSIS

The following methods of statistical analysis have been used in this study. The results were averaged (mean±standard deviation) for continuous data, and number and percentage for dichotomous data are presented in Table and Figure.

1. One way Analysis of Variance (ANOVA)

One way analysis of variance was used to test the difference between groups. Analysis of Variance is a technique by which the total variation is split into two parts one between groups and the other within the groups. If 'F' value is significant, there is a significant difference between group means. To find out which of the two groups means is significantly different post hoc test of LSD test is used. In case F value is not significant it indicates that there is no significant difference between the groups and stops the analysis at this stage and does not use LSD test.

The formula used: where MS=Mean Sum of Square

2. Univariate analysis of the dichotomous variables encoded was performed

By means of the Chi square test with Yates correction if required.

Chi-Square (γ2) test for (2 x 2 tables)

Group	**Attribute Characteristic finding**		**Total**
	Absent	**Present**	
Male	a	b	a+b
Female	c	d	c+d
Total	a+c	b+d	N

a, b, c, d are the observed numbers.

N is the Grand Total **γ**2 with 1 DF

DF=(r-1)*(c-1), where r=rows and c=columns

DF= Degrees of Freedom (Number of observation that are free to vary after certain restriction have been placed on the data).

3. **Student "t' test**

The student 't' test was used to determine whether there was a statistical difference between male and female subjects in the parameters measured. Student's t test is as follows: t= ~~~ t n1+n2-2

In all the above tests, p values less than 0.05 were taken to be statistically significant. The data was analyzed using SPSS package.

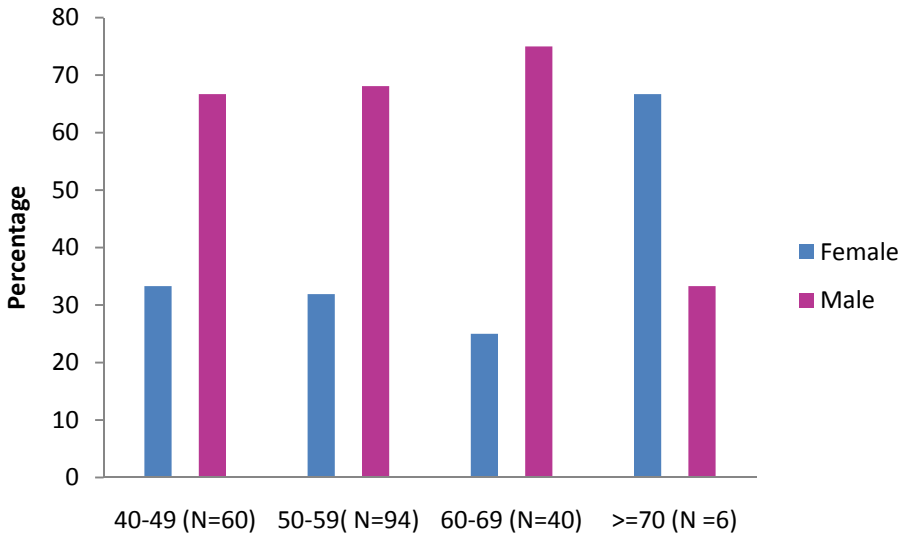
RESULTS

200 diabetic patients with 64 (32.0%) females and 136 (68.0%) males were included in the study. All the subjects were studied in terms of age, IOP, duration of diabetics and blood glucose level. The observations were made in both eyes of all the subjects.

Table 1 Age and sex distribution of diabetics in the study group

Age group	**Sex**		**Total**
	Female (%)	**Male (%)**	
40-49	**20**(33.3)	**40**(66.7)	**60**
50-59	**30**(31.9)	**64**(68.1)	**94**
60-69	**10**(25.0)	**30**(75.0)	**40**
>=70	**4(66.7)**	**2(33**.3)	**06**
Total	**64**	**136**	**200**
Statistic	**DF**	**Value**	**Prob**
Chi-Square	**3**	2.1319	0.5455

Fig 1: Age and Gender distribution of the Study Population



The study population consists of 136 (68.0%) males and 64(32.0%) females in the age range from 40-73 years with mean age of 53.1±8.28 years. The total number of diabetic patients observed in 40 to 49 years is 60 (30.0%), 50 to 59 years 94(47.0%), 60-69 years 40 (20.0%) and >70 years 6 (3.0%). The majority of the diabetics are above 50 years age. The age specific diabetics in males was 66.7% (40 out of 60) in the age group of 40 to 49 years followed by 68.1% (64 out of 94) in the age group 50 to 59 years, 75.0% (30 out of 40) in 60-69 years and 33.3% (2 out of 6) in >70 years. The age specific diabetics in females was 33.3% (20 out of 60) in the age group of 40 to 49 years followed by 31.9% (30 out of 94) in the age group 50 to 59 years, 25.0% (10 out of 40) in 60-69 years and 66.7%(4 out of 6) in >70 years.

The table shows the majority of diabetic patients in the study are above 50 years (70%). The difference observed was not statistically significant (p>0.05). Age and sex distribution was similar in the study.

Fig 2: Gender distribution among the Study population

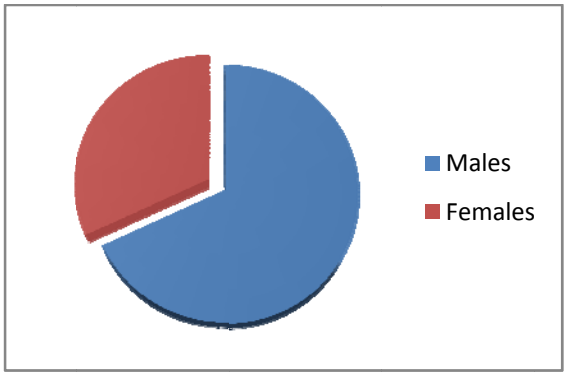


Table 2-Sex-wise distribution of patients with POAG

Sex	**Total No. of patients**	**Diagnosed**			
		POAG (%)	**NTG (%)**	**OH (%)**	**Normal (%)**
Males	**136**	**7**(5.1)	**4**(2.9)	**0**	**125**(91.9)
Females	**64**	**2**(3.1)	**0**	**2**(3.1)	**60**(93.8)
Total	**200**	**9**(4.5)	**4**(2.0)	**2**(1.0)	**185**(92.5)

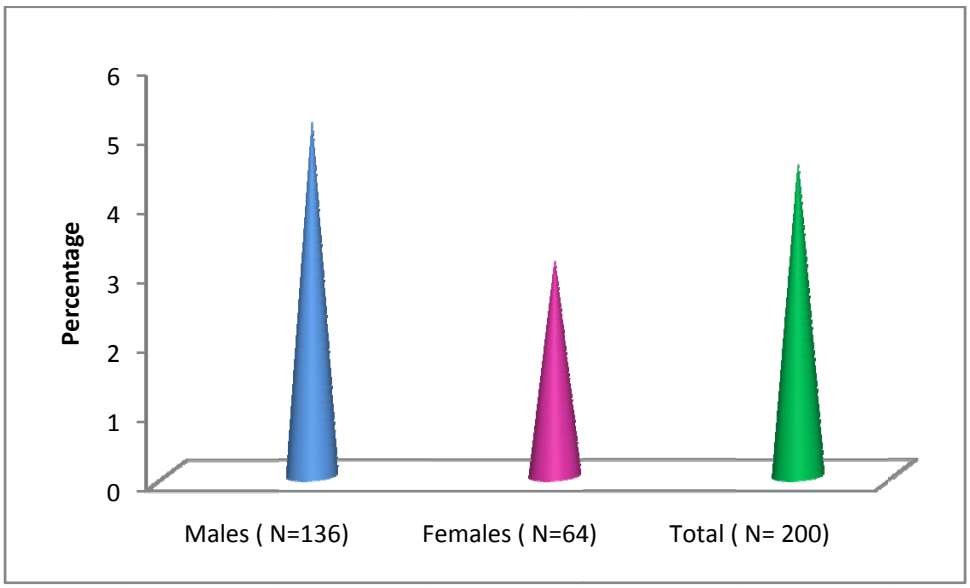
This table shows the proportion of cases diagnosed among diabetic patients in the study population. Overall proportion of POAG cases observed was 4.5% (9 out 200), NTG observed 2.0% (4 out of 200) and 1.0% OH cases were observed remaining 93% were normal. Among the males 5.1% (7 out of 136) and females 3.12% (2 out 64) POAG cases were diagnosed. The proportion of POAG cases diagnosed between males and females was not statistically significant ($p>0.05$) as shown in the following table 3 .

Table 3 : Sex distribution

Sex	POAG			
	Present		Present	
	No	%	No	%
Male	7	5.1	129	94.9
Female	2	3.1	62	96.9
Total	9	4.5	191	95.5
'p'	0.4069 Not significant			

5.1% of the males and 3.1% of the females had POAG in this study.. The sex composition of the two groups was not significantly different (p = 0.4069).

Fig 3: Gender wise distribution of POAG patients



RELATIONSHIP BETWEEN PREVALENCE OF POAG AND OTHER VARIABLES IN DIABETIC CASES

Table 4 :

Age group	POAG			
	Present		Absent	
	No	%	No	%
< 50 years (60)	-	-	60	100
50- 59 years (94)	6	6.4	88	93.6
60-69 years (40)	2	5.0	38	95
≥ 70 yrs (6)	1	16.7	5	83.3
Total	9	4.5	191	95.5
Mean	56.2		52.8 years	
SD	6.6 years		8.5 years	
'p'	0.2826 Not significant			

The patients with POAG had an age of 56.2 $\pm$6.6 years. The cases without POAG had an age of 52.8 $\pm$8.5 years. There was no statistically significant difference in the age composition of the two groups. ($p > 0.05$).

Table 8 -Age-wise distribution of POAG among the diabetic population studied

Age group	Male	Female	Total
40-49	-	-	-
50-59	**4**	**2**	**6**
60-69	**2**	-	**2**
>=70	**1**	-	-
Total	**7**	**2**	**9**

The above table shows the age distribution among POAG patients (9 out of 200). The mean age of the patients was 54.5±4.80 ranging from 50 yrs to 61 yrs. The mean age of male patients was 56.0± 4.58 ranging from 52 yrs to 61 yrs. The mean age of female patients was 50.0 years.

Table 9 - IOP among patients with POAG

Age	Sex	IOP (mmHg)	
		RE	LE
55	Male	**31.8**	**25.1**
50	Female	**23.8**	**22.4**
52	Male	**25.1**	**22.4**
61	Male	**23.1**	**25.1**
50	Female	**23.8**	**22.4**
61	Male	**23.1**	**25.1**
55	Male	**31.8**	**25.1**
52	Male	**25.1**	**22.4**
70	Male	**22.4**	**22.4**

The above table shows the IOP values of Right and left eye among POAG Cases.

Table 10 : IOP and prevalence of POAG in DM cases

IOP	POAG				'p'
	Present		Absent		
	Mean	S.D.	Mean	S.D.	
Right Eye	27.49	4.17	16.62	2.62	**0.0001 Significant**
Left Eye	25.82	4.98	17.82	2.6	**0.0001 Significant**

IOP in right eyes were (27.49 ± 4.17) significantly higher for patients with POAG than for cases without POAG (16.62 ± 2.62). Similarly in left eye also IOP values were significantly higher for POAG cases.

Thus there was statistically significant association between IOP values and incidence of POAG.('p' = 0.0001)

Fig 4: Comparison of mean intraocular pressure (Right Eye) among normal patients and those diagnosed

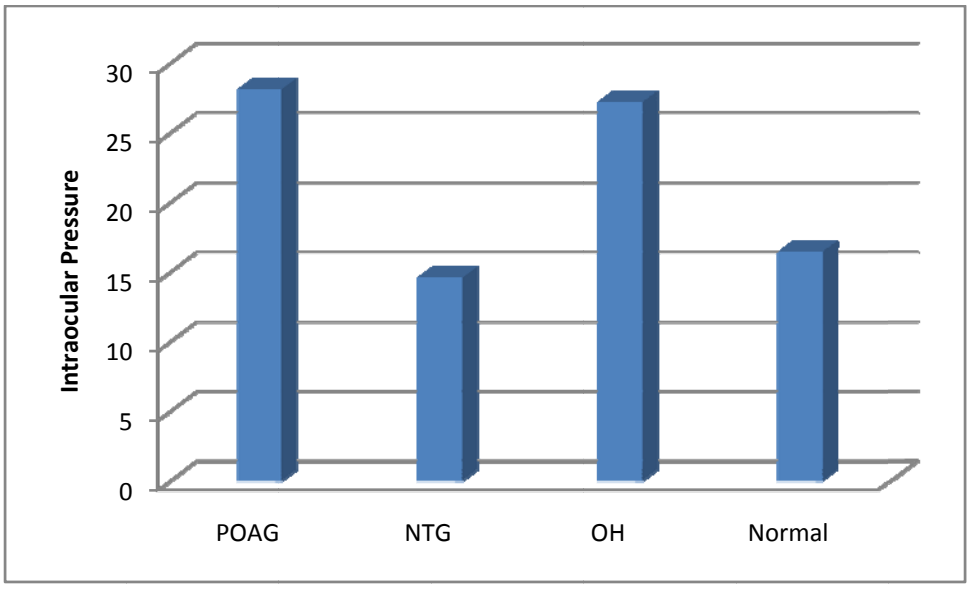
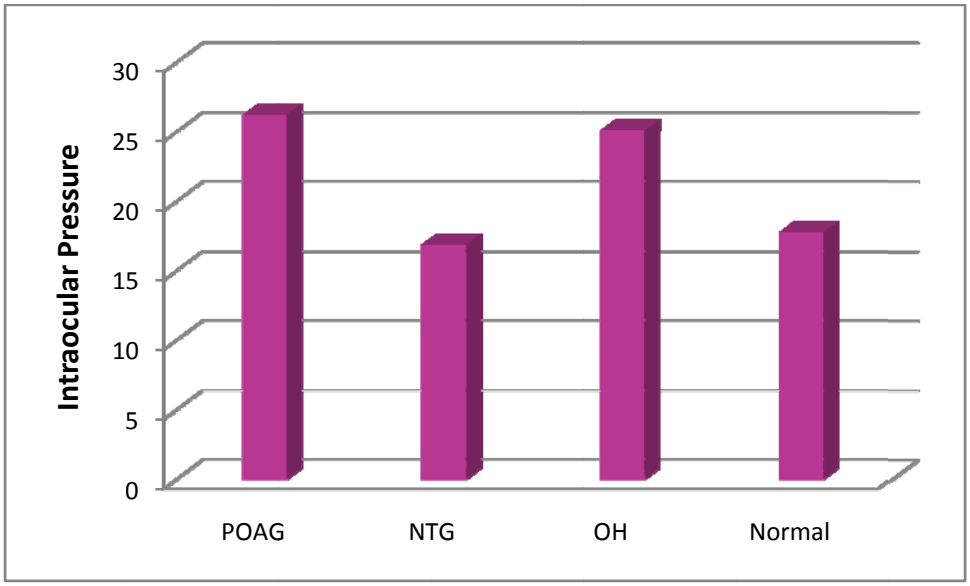


Fig 5: Comparison of mean intraocular pressure (Left Eye) among normal patients and those diagnosed



Those patients with IOP ≤ 21 mmHg with glaucomatous disc damage, visual field loss and open angle on gonioscopy were suspected to have normal tension glaucoma. Those patients with IOP > 21 mmHg with no disc changes and no visual field defects were suspected to have ocular hypertension.

The Mean Right eye IOP values of Normal were 16.46 ± 2.43. The mean IOP values of NTG were 14.60 ± 0.65. The mean IOP values of POAG were 27.49 ± 4.17. The mean IOP values of OH were 27.2. The difference observed in mean IOP values among the group was statistically significant ($p<0.05$).

The further analysis of comparison of mean IOP between the groups (initial diagnosis) revealed that the NTG patients had statistically significant

lower IOP than POAG and OH patients ($p<0.05$) but no significant difference is seen between NTG and Normal ($p>0.05$). The POAG patients had statistically significant higher IOP than Normal.

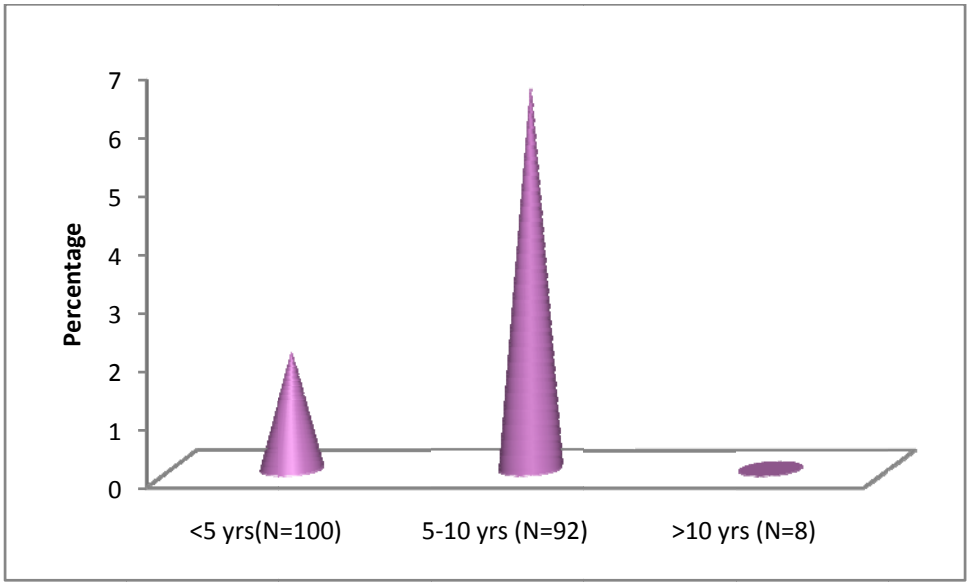
The Mean Left eye IOP values of Normal were 17.79±2.55. The mean IOP values of NTG were 16.9±0.57. The mean IOP values of POAG were 25.82 $\pm$4.98. The mean IOP values of OH were 25.1. The difference observed in mean IOP values among the group was statistically significant ($p<0.05$).

The further analysis of comparison of mean IOP between the groups (initial diagnosis) revealed that the NTG patients had statistically significant lower IOP than POAG and OHT patients ($p<0.05$) but no significant difference is seen between NTG and Normal ($p>0.05$). The POAG patients had statistically significant higher IOP than Normal.

Table 11 - Distribution of POAG cases according to duration of DM

Duration of DM	No. of Patients	POAG	Percentage
<5 yrs	**100**	**02**	**2**
5-10 yrs	**92**	**06**	**6.52**
>10 yrs	**08**	**-**	**--**
Total	**200**	**08**	**4.00**

Fig 6: Distribution of POAG cases according to duration of DM



The above table shows the duration of diabetes among POAG patients. It was observed that 75%(6 out 8) were suffering from diabetes between 5 to 10 years and 25% (2 out 8) were <5 years suffering from diabetes for 5 years.

Table 7- Age distribution of patients diagnosed

Age group	Diagnosis				Total
	POAG	NTG	OH	N	
40-49	**0**	**0**	**0**	**60**	**60**
50-59	**6**	**4**	**0**	**84**	**94**
60-69	**2**	**0**	**2**	**36**	**40**
>=70	**1**	**0**	**0**	**3**	**03**
Total	**9**	**4**	**2**	**185**	**200**

The study population consists of 136 (68.0%) male and 64(32.0%) female in the age range from 40-73 years with mean age of 53.1±8.28 years. The total number of diabetic patients observed in 40 to 29 years is 60 (30.0%), 50 to 59 years 94(47.0%), 60-69 years 40 (20.0%) and >70 years 6 (3.0%). The majority of the diabetics are above 50 years age.

The table shows that the proportion of POAG is more in patients above 50 years age. It also shows the prevalence of NTG is more in patients in the age group of 50-59 years.

Table 8- Comparison of mean blood glucose level among diabetics with POAG and without POAG

Mean blood glucose level	Diagnosis		t value	p value
	POAG	Others		
FBS	200.25	115.51	4.21	<0.01
PPBS	305.75	198.41	3.65	0.0004

Fig 7: Comparison of mean blood glucose level among diabetics with POAG and without POAG

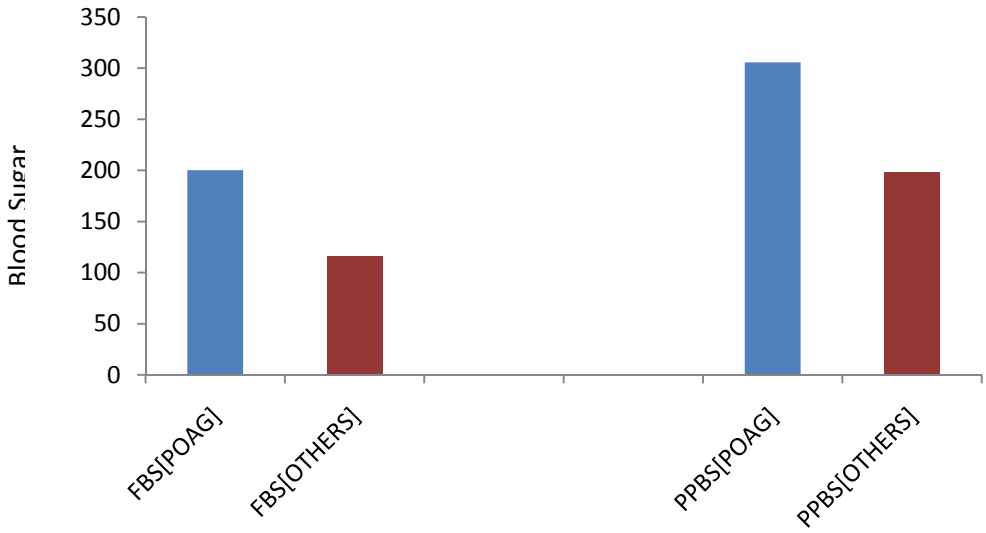
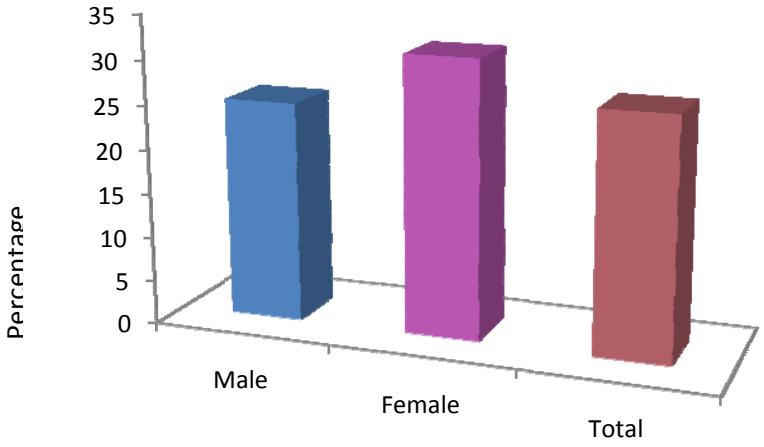


Table 9- Distribution of proportion of Diabetic Retinopathy among diabetic patients

Sex	No of pts	DR	Percentage
Male	**136**	**34**	**25.0**
Female	**64**	**20**	**31.3**
Total	**200**	**54**	**27.0**
Statistic	**DF**	**Value**	**Prob**
Chi-Square	**1**	0.4313	0.5114

Fig 8: Distribution of proportion of Diabetic Retinopathy among diabetic patients



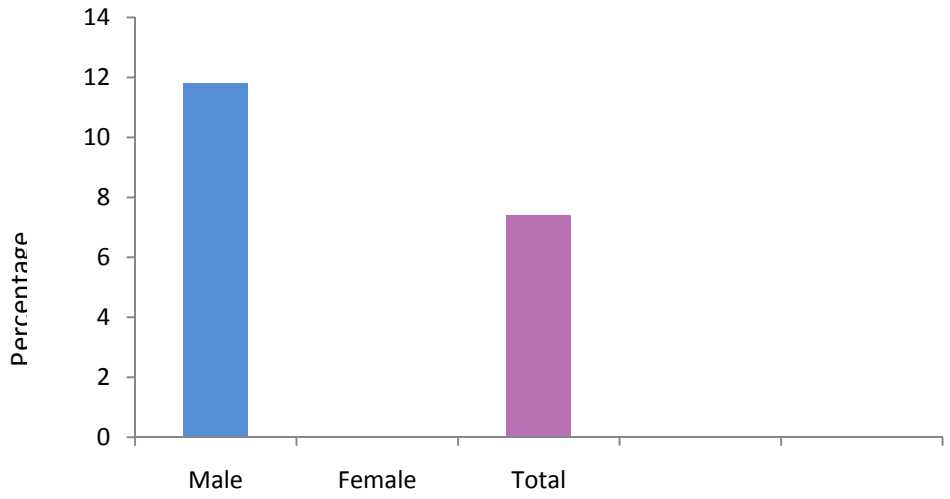
The mean blood glucose level (FBS) was 200.25 among POAG and 115.51 among other patients. The mean difference observed was statistically significant (p=0.05). The PPBS level among POAG was 305.75 and among others was 198.41. The difference observed was statistically significant (p<0.05). The mean blood glucose level is higher in diabetics with POAG than others.

Table 10- Proportion of POAG among Diabetic patients with retinopathy

Sex	DR	POAG	Percentage
Male	**34**	**4**	**11.8**
Female	**20**	**0**	**0**
Total	**54**	**4**	**7.4**

The above table shows the proportion of POAG among those having diabetic retinopathy. Overall 7.4% (4 out 54) POAG patients had diabetic retinopathy. Among males it was observed that 11.8% (4 out of 34) were diagnosed as POAG, and among females no patients were diagnosed as POAG. It was observed that proportion was more in male diabetics than in females.

Fig 9: Proportion of POAG among Diabetic patients with Retinopathy



This table shows the proportion of Diabetic retinopathy among diabetic patients in the study population. Overall proportion of cases observed was 27.0% (54 out 200). Among the males 25.0% (34 out of 136) and females 31.3% (20 out 64) cases were observed. The proportion of Diabetic retinopathy observed between males and females was not statistically significant ($p>0.05$).

Statistical Tools

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using **Epidemiological Information Package (EPI 2010)** developed by Centre for Disease Control, Atlanta.

Using this software range, frequencies, percentages, means, standard deviations, chi square and 'p' values were calculated. Kruskul Wallis chi-square test was used to test the significance of difference between quantitative variables and Yate's chi square test for qualitative variables. A 'p' value less than 0.05 is taken to denote significant relationship.

DISCUSSION

The discussion of an association between Diabetes and POAG is not new. In 1971 Becker stated “Diabetes Mellitus occurs more often in patients with Primary Open Angle Glaucoma than in non-glaucomatous populations. Similarly, Glaucoma is more prevalent in diabetic than in non-diabetic population”.Considerable controversy exists in literature.

While several studies show an association between the two diseases, several others fail to show any significant association. Most of these studies were comparatively small, used differing definitions of glaucoma and were clinical, rather than community based. A prevalence of 3.11 from Rotterdam, 1.84 from Wisconsin and 2.12 from Australia have been reported. Armstrong et al have reported a prevalence of POAG of 4.1 % in diabetic patients. The prevalence of diabetes in POAG is 1.7 %. A community based study conducted in Vellore, South India showed a prevalence of POAG of 4.1% in diabetics.

My study shows a clear evidence of an excess of POAG in diabetic population, which is 4.5 % .The prevalence among males, is slightly more (5.1 %) as compared to females (3.12%). In my study, the mean age of POAG among males was 54.5 yrs and 50.0 yrs among females. My studies show that the prevalence of POAG and the duration of DM is proportional and that the

mean blood glucose level is higher in diabetics with POAG. Screening all diabetic patients for POAG at the time of annual screening for retinopathy is an attractive proposition provided a clear clinical benefit could be demonstrated. A screening test should ideally be relatively inexpensive, simple, and quick to perform and if possible be capable of being administered by a nonspecialist.

SUMMARY

Primary Open Angle Glaucoma is characterized by its adult onset, IOP > 21mm Hg at some point in the course of the disease, open normal angle, glaucomatous optic nerve damage and visual field loss. It affects 1 in 100 people above 40 years of age. Diabetes Mellitus is one of the risk factors for POAG along with others such as positive family history, high myopia, black race etc. Various studies have addressed this question with some studies upholding and some refuting a link between the two diseases. Studies among Asians are limited and most studies are population based. In my study I found a significant association between POAG and Diabetes. I found a moderate excess of prevalence of POAG among patients with diabetes and that this was proportional to the duration of Diabetes. These findings are compatible with studies that have demonstrated an association between the two diseases.

Primary Open Angle glaucoma is typically asymptomatic until significant visual field loss has occurred. Patients usually present with significant visual field loss in one eye and advanced disease in the other. It is clearly evident from my study that diabetic patients are indeed at significant risk of developing primary open angle glaucoma, and hence screening is very important.

CONCLUSION

1. Primary Open Angle glaucoma is typically asymptomatic until significant visual field loss has occurred.
2. Patients usually present with significant visual field loss in one eye and advanced disease in the other.
3. It is associated with irreversible blindness.
4. Thus, the public health importance of detecting undiagnosed and treatable glaucoma is important, as blindness has economic and societal consequences for the rest of an individual's life.
5. Several studies have shown an association between POAG and diabetes.
6. From my study, I come to a conclusion that there is an excess of POAG in diabetic population, which is 4.5% (as compared to 2.1% in normal population), thereby showing an association between primary open angle glaucoma and diabetes.

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PROFORMA

A CLINICAL STUDY OF PRIMARY OPEN ANGLE GLAUCOMA IN DIABETIC PATIENTS

PERSONAL DATA

Name:

• Age:

• Sex:

• Address:

• Presenting Complaints:

• History of presenting complaints:

• Medical history:

• Past history:

• Treatment history:

• Family history:

• Personal history:

• Duration of DM:

GENERAL PHYSICAL EXAMINATION

SYSTEMIC EXAMINATION

OCULAR EXAMINATION:

1. Visual acuity RE

LE

2. External ocular examination

• Head posture

• Facial Symmetry

RE LE

• Eyeball

• Conjunctiva

• Sclera

• Cornea

• Anterior Chamber

• Iris

• Pupil

• Lens

3. Fundus Examination

• Media

• Optic disc

• Macula

• Retinal blood vessels

• Background

4. Diagnostic tests

a. Tonometry (by Schiotz indendation tonometer)

b. Slit Lamp Examination

c. Gonioscopy

d. Perimetry (by Octopus 300 field analyser)

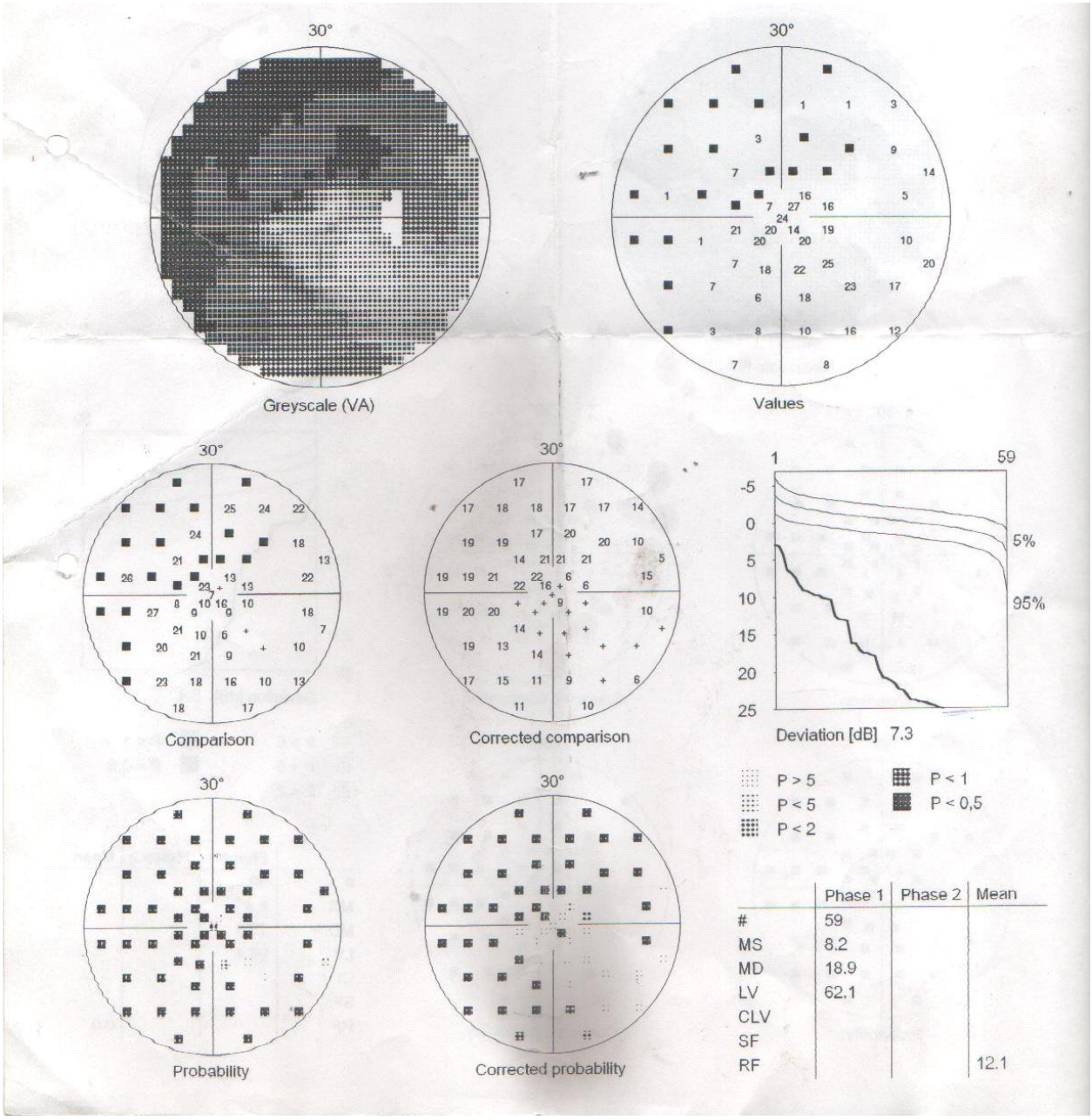
FBS and PPBS

INFERENCE

POAG - (BE)

Imp: Supra with Arcuate Scotoma

HAAG-STREIT INTERNATIONAL OCTOPUS 300 V5.17
Seven-in-One



	Phase 1	Phase 2	Mean
#	59		
MS	8.2		
MD	18.9		
LV	62.1		
CLV			
SF			
RF			12.1

KEY TO MASTER CHART

Sl. No. → Serial number

OP.No. → Outpatient number

M → Male

F → Female

DM → Diabetes Mellitus

yrs → Years

RE → Right Eye

LE → Left Eye

IOP → Intraocular pressure

A.S → Anterior Segment

N → Normal

A → Absent

P → Present

DR → Diabetic Retinopathy

Glau. → Glaucomatous

Gonio → Gonioscopy

FBS → Fasting Blood Sugar

PPBS → Postprandial blood sugar

POAG → Primary open angle Glaucoma

NTG → Normal Tension Glaucoma

OH → Ocular Hypertension

MASTER CHART

Sl No	Name	OP No	Age	Sex	Duration of diabetes {yrs}	Vision RE	Vision LE	IOP RE mm hg	IOP LE mm hg	AS	Disc changes	DR	Glau field changes	Gonio	FBS/PPBS	POAG	NTG	OH
1	Selvaraj	470235	62	M	8	6/9	6/18	14.6	18	N	A	A	A	IV	96/152	A	A	A
2	Ramaraj	470300	65	M	7	6/9	6/18	14.6	18	N	A	P	A	IV	98/160	A	A	A
3	Devaraj	470302	50	M	4	6/6	6/18	18	17.3	N	A	A	A	IV	88/139	A	A	A
4	VarisaiMuthu	470324	58	M	5	6/9	6/18	17.3	17.3	N	A	P	A	IV	108/187	A	A	A
5	Nagarajan	470326	69	M	6	6/9	6/12	17.3	18	N	A	A	A	IV	88/150	A	A	A
6	Sekar	470356	55	M	1	6/6	6/9	17.3	17.3	N	A	A	A	IV	129/230	A	A	A
7	Murugesan	470328	54	M	2	6/9	6/9	14.6	17.3	N	A	P	A	IV	110/186	A	A	A
8	Mariappan	470330	58	M	4	6/9	6/9	14.6	17.3	N	A	A	A	IV	102/185	A	A	A

9	Yamuna	470540	65	F	5	6/9	6/9	27.2	25.1	N	A	A	A	IV	110/240	A	A	P
10	Krishnaraj	470402	60	M	6	6/24	6/18	17.3	16.5	N	A	A	A	IV	98/160	A	A	A
11	Muthurajan	470499	57	M	5	6/6	6/6	17.3	17.3	N	A	P	A	IV	98/201	A	A	A
12	Chandran	470678	65	M	9	6/12	6/12	17.3	17.3	N	A	A	A	IV	98/160	A	A	A
13	Gangamuthu	470699	61	M	5	6/36	6/36	17.3	17.3	N	A	A	A	IV	114/206	A	A	A
14	Karuppaiah	470701	62	M	7	6/12	6/18	17.3	17.3	N	A	A	A	IV	98/168	A	A	A
15	Samsudeen	470702	60	M	8	6/18	6/36	17.3	17.3	N	A	P	A	IV	100/210	A	A	A
16	Radhakrishnan	480788	59	M	3	6/6	6/6	17.3	18	N	A	A	A	IV	88/150	A	A	A
17	Kundumuthu	470783	52	M	6	6/18	6/12	31.8	34.4	N	P	A	P	IV	20/273	P	A	A
18	Bose	470790	54	M	1	6/9	6/9	17.3	15.1	N	A	A	A	IV	98/160	A	A	A
19	Hajira Biwi	470792	52	F	1	6/9	6/12	12.6	15.1	N	A	A	A	IV	88/139	A	A	A
20	Vadakarayan	470794	52	M	4	6/9	6/9	17.3	17.3	N	A	A	A	IV	110/18	A	A	A

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21	Muthupandi	470785	59	M	5	6/9	6/9	17.3	17.3	N	A	A	A	IV	100/210	A	A	A
22	Manikandan	470820	64	M	7	6/18	6/18	17.3	17.3	N	A	P	A	IV	106/211	A	A	A
23	Pounraj	470824	62	M	5	6/18	6/18	20.6	18	N	A	A	A	IV	100/198	A	A	A
24	Munusamy	470826	57	M	3	6/9	6/9	14.6	17.3	N	A	A	A	IV	88/150	A	A	A
25	Mohammed Aysha	470888	51	F	2	6/9	6/9	14.6	17.3	N	A	A	A	IV	96/182	A	A	A
26	Mohammed Ismail	470890	64	M	5	6/12	6/12	17.3	20.6	N	A	A	A	IV	76/142	A	A	A
27	Govindarajan	470891	60	M	5	6/12	6/12	18	20.6	N	A	A	A	IV	110/186	A	A	A
28	Poosari	470887	55	M	1	6/9	6/9	14.6	17.3	N	A	A	A	IV	112/198	A	A	A
29	Natarajan	470920	40	M	0.1	6/6	6/6	14.6	17.3	N	A	A	A	IV	88/139	A	A	A
30	Mookammal	470931	55	F	7	6/9	6/12	16.5	17.3	N	A	P	A	IV	110/187	A	A	A
31	Thirupathi	470945	55	M	7	6/9	6/9	14.6	17.3	N	A	P	A	IV	100/222	A	A	A
32	Parasuraman	47095	40	M	0.6	6/6	6/6	14.	17.	N	A	A	A	IV	96/160	A	A	A

		0						6	3											
33	Veerannan	47095 7	53	M	4	6/12	6/12	13.8	15.1	N	A	A	A	IV	129/230	A	A	A
34	Sellandi	47095 8	53	M	5	6/9	6/9	13.8	15.1	N	A	A	A	IV	88/146	A	A	A
35	Solaimalai	47095 5	42	M	0.6	6/6	6/9	15.1	13.8	N	A	A	A	IV	108/187	A	A	A
36	Devaraj	47097 0	61	M	6	6/6	6/6	25.1	23.1	N	P	P	P	IV	106/260	P	A	A
37	Theen Fathima	47097 5	55	F	8	6/9	6/9	15.1	14.6	N	A	P	A	IV	98/160	A	A	A
38	Pichayappan	47097 7	55	M	6	6/60	6/36	14.6	12.6	N	A	P	A	IV	110/118	A	A	A
39	Muthuvel	47098 2	42	M	1	6/18	6/18	14.6	12.6	N	A	A	A	IV	112/180	A	A	A
40	Nageswari	47098 3	50	F	4	6/9	6/9	17.3	17.3	N	A	A	A	IV	86/160	A	A	A
41	Sathappan	47098 7	50	M	6	6/9	6/12	14.7	17.3	N	A	A	A	IV	120/170	A	P	A
42	Subramani	47099 0	55	M	6	6/18	6/12	13.8	14.6	N	A	A	A	IV	78/140	A	A	A
43	Abdul Khader	47099 5	42	M	1	6/6	6/6	12.6	15.1	N	A	A	A	IV	88/139	A	A	A
44	Palani	47100 3	66	M	7	6/1	6/2	17.3	18	N	A	P	A	IV	97/189	A	A	A

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45	Sivasami	471020	59	M	7	6/12	6/12	15.1	15.1	N	A	P	A	IV	96/180	A	A	A
46	Perumal	471030	51	M	1	6/12	6/12	12.6	15.1	N	A	A	A	IV	100/176	A	A	A
47	Ganapathy	471032	40	M	0.2	6/36	6/24	14.6	17.3	N	A	A	A	IV	92/150	A	A	A
48	Karunakaran	471050	58	M	3	6/9	6/9	14.6	17.3	N	A	A	A	IV	129/200	A	A	A
49	Silayappan	471053	42	M	1	6/24	6/24	12.6	14.6	N	A	A	A	IV	88/164	A	A	A
50	Parvathy	471057	56	F	5	6/12	6/12	15.1	14.6	N	A	A	A	IV	96/150	A	A	A
51	Aandal	471066	40	F	0.6	6/12	6/12	13.8	15.1	N	A	A	A	IV	111/180	A	A	A
52	Meenatchi	471078	43	F	1	6/9	6/9	14.6	17.3	N	A	A	A	IV	126/222	A	A	A
53	Krishnasamy	471082	45	M	2	6/9	6/9	14.6	17.3	N	A	A	A	IV	140/312	A	A	A
54	Kannan	471085	41	M	1	6/18	6/12	17.3	20.6	N	A	A	A	IV	88/150	A	A	A
55	Sasidharan	471089	50	M	3	6/18	6/12	12.6	15.1	N	A	A	A	IV	76/136	A	A	A
56	Abdul Malik	47109	49	M	2	6/9	6/9	17.	20.	N	A	A	A	IV	129/23	A	A	A

		3						3	6						0				
		47109						23.	22.						225/30				
57	Malathi	5	50	F	0.6	6/6	6/6	8	4	N	P	A	P	IV	7	P	A	A	
		47110						20.	22.						110/19				
58	Ganga	1	53	F	6	6/6	6/6	6	4	N	A	A	A	IV	6	A	A	A	
		47112						22.	22.										
59	Sekkadiyan	0	56	M	5	6/6	6/6	4	4	N	A	A	A	IV	76/150	A	A	A	
		47112						17.	20.						160/32				
60	Amaravathy	3	58	F	2	6/6	6/6	3	6	N	A	A	A	IV	0	A	A	A	
		47112						20.	17.						100/18				
61	Santhanam	5	41	M	2	6/6	6/6	6	3	N	A	A	A	IV	0	A	A	A	
		47113						17.	14.						187/32				
62	Ramasamy	6	56	M	7	6/9	6/9	3	6	N	A	A	A	IV	0	A	A	A	
		47113						15.	17.										
63	Periyasamy	8	50	M	4	6/9	6/9	1	3	N	A	A	A	IV	92/178	A	A	A	
		47113						20.	22.										
64	Sivankalai	9	50	M	5	6/9	6/9	6	4	N	A	A	A	IV	89/162	A	A	A	
		47115				6/2	6/1	22.	22.						208/28				
65	Muthuramalingam	0	70	M	8	4	8	4	4	N	P	P	P	IV	7	P	A	A	
		47115				6/2	6/2	17.	17.						110/18				
66	Paapammal	2	65	F	20	4	4	3	3	N	A	A	A	IV	6	A	A	A	
		47116						14.	17.										
67	Narayanan	2	40	M	1.5	6/6	6/6	6	3	N	A	A	A	IV	68/142	A	A	A	
		47120				6/1		14.	16.						167/24				
68	Kadhiresan	1	50	M	3	2	6/9	6	5	N	P	P	P	IV	1	A	P	A	
		47125						14.	14.										
69	Nagadevan	0	44	M	1	6/6	6/6	6	6	N	A	A	A	IV	84/160	A	A	A	
		47126						14.	17.										
70	Jamruth Begum	1	50	F	2	6/1	6/1	6	3	N	A	A	A	IV	88/158	A	A	A	

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71	Magamayi	471267	52	F	8	6/9	6/9	17.3	17.3	N	A	A	A	IV	104/186	A	A	A
72	Balakrishnan	471274	48	M	8	6/18	6/60	22.4	23.8	N	A	P	A	IV	96/150	A	A	A
73	Chinnathayi	471277	46	F	0.1	6/6	6/6	20.6	20.6	N	A	A	A	IV	135/270	A	A	A
74	Janaki	471283	52	F	8	6/6	6/6	17.3	20.6	N	A	P	A	IV	96/170	A	A	A
75	Shanthanagu	471298	56	M	4	6/18	6/36	17.3	16.5	N	A	A	A	IV	120/198	A	A	A
76	Rajathi	471232	45	F	7	6/6	6/6	20.6	22.4	N	A	A	A	IV	120/186	A	A	A
77	Nagajothi	471267	48	F	10	6/9	6/6	20.6	20.6	N	A	P	A	IV	118/190	A	A	A
78	Kasthuri	471284	46	F	0.6	6/9	6/9	20.1	23.8	N	A	P	A	IV	130/269	A	A	A
79	Mookammal	471293	40	F	4	6/6	6/6	17.3	17.3	N	A	A	A	IV	110/170	A	A	A
80	Badrinathan	481321	55	M	10	6/18	6/18	31.8	25.1	N	P	P	P	IV	270/383	P	A	A
81	Afsar Urusen	471345	48	M	4	6/18	6/12	15.9	15.9	N	A	P	A	IV	96/142	A	A	A
82	Pushpa Valli	471390	40	F	1	6/6	6/6	17.3	17.3	N	A	A	A	IV	85/199	A	A	A
83	Rajalakshmi	471394	45	F	1	6/18	6/18	17.3	17.3	N	A	A	A	IV	129/230	A	A	A

84	Ramachandran	471401	40	M	4	6/6	6/6	15.9	15.9	N	A	A	A	IV	167/226	A	A	A
85	Panjavarnam	471453	58	F	0.1	6/9	6/9	14.6	20.6	N	A	A	A	IV	170/300	A	A	A
86	Jyothikrishna	471458	50	M	8	6/6	6/6	14.6	17.3	N	A	A	A	IV	96/140	A	A	A
87	Banu	471505	55	F	8	6/12	6/12	14.6	20.6	N	A	P	A	IV	110/186	A	A	A
88	Muthiah	471529	65	M	10	6/6	6/6	14.6	14.6	N	A	A	A	IV	102/193	A	A	A
89	Rajendran	471537	40	M	6	6/6	6/6	14.6	14.6	N	A	A	A	IV	160/220	A	A	A
90	Velrajammal	471546	73	F	20	6/9	6/12	14.6	20.6	N	A	A	A	IV	116/186	A	A	A
91	Krishnaveni	471678	60	F	27	6/18	6/18	20.6	20.6	N	A	P	A	IV	114/190	A	A	A
92	Lakshmi	471673	70	F	0.5	6/9	6/9	17.3	17.3	N	A	P	A	IV	100/202	A	A	A
93	Kuruvammal	471543	44	F	2	6/6	6/6	20.6	22.4	N	A	N	A	IV	79/178	A	A	A
94	Gopal	471790	51	M	5	6/12	6/12	17.3	24.4	N	A	P	A	IV	80/160	A	A	A
95	Ramalingam	471793	48	M	2	6/6	6/6	17.3	17.3	N	A	A	A	IV	100/168	A	A	A
96	Kasiammal	471795	64	F	15	6/18	6/18	14.6	17.3	N	A	P	A	IV	140/220	A	A	A
97	Panju	47182	65	F	10			20.	20.	N	A	A	A	IV	110/17	A	A	A

		0				6/60	6/12	6	4						5					
98	Sundaresan	481837	45	M	6	6/12	6/12	20.6	20.6	N	A	A	A	IV	120/186	A	A	A		
99	Seethayammal	471844	52	F	3	6/6	6/6	20.6	20.6	N	A	P	A	IV	110/186	A	A	A		
100	Easwaran	471852	32	M	3	6/9	6/9	14.6	17.3	N	A	A	A	IV	76/110	A	A	A		
101	Dhanapal	471902	62	M	8	6/9	6/18	14.6	18	N	A	A	A	IV	96/152	A	A	A		
102	Kondayan	471920	65	M	7	6/9	6/18	14.6	18	N	A	P	A	IV	98/160	A	A	A		
103	Paraman	471999	50	M	4	6/6	6/18	18	17.3	N	A	A	A	IV	88/139	A	A	A		
104	Pandian	482003	58	M	5	6/9	6/18	17.3	17.3	N	A	P	A	IV	108/187	A	A	A		
105	Pasupathy	472006	69	M	6	6/9	6/12	17.3	18	N	A	A	A	IV	88/150	A	A	A		
106	Senthil	472098	55	M	1	6/6	6/9	17.3	17.3	N	A	A	A	IV	129/230	A	A	A		
107	Balaraman	472108	54	M	2	6/9	6/9	14.6	17.3	N	A	P	A	IV	110/186	A	A	A		
108	Palpandi	472200	58	M	4	6/9	6/9	14.6	17.3	N	A	A	A	IV	102/18	A	A	A		

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109	Podhumponnu	472285	65	F	5	6/9	6/9	27.2	25.1	N	A	A	A	IV	110/240	A	A	P
110	Paramaguru	472504	60	M	6	6/4	6/18	17.3	16.5	N	A	A	A	IV	98/160	A	A	A
111	Veeranna Thevar	502618	57	M	5	6/6	6/6	17.3	17.3	N	A	P	A	IV	98/201	A	A	A
112	Kumaravel	472620	65	M	9	6/12	6/12	17.3	17.3	N	A	A	A	IV	98/160	A	A	A
113	Muthusamy	472780	61	M	5	6/36	6/36	17.3	17.3	N	A	A	A	IV	114/206	A	A	A
114	Azhagarsamy	472890	62	M	7	6/12	6/18	17.3	17.3	N	A	A	A	IV	98/168	A	A	A
115	Senthamarai	472912	60	M	8	6/18	6/36	17.3	17.3	N	A	P	A	IV	100/210	A	A	A
116	Mayilsamy	472918	59	M	3	6/6	6/6	17.3	18	N	A	A	A	IV	88/150	A	A	A
117	Rahavan	473056	52	M	6	6/18	6/12	31.8	34.4	N	P	A	P	IV	20/273	P	A	A
118	Balaji	473270	54	M	1	6/9	6/9	17.3	15.1	N	A	A	A	IV	98/160	A	A	A
119	Rani	473430	52	F	1	6/9	6/12	12.6	15.1	N	A	A	A	IV	88/139	A	A	A
120	Mannaru	473504	52	M	4	6/9	6/9	17.3	17.3	N	A	A	A	IV		A	A	A

0		4						3	3						110/188				
121	Rajappan	473682	59	M	5	6/9	6/9	17.3	17.3	N	A	A	A	IV	100/210	A	A	A	
122	Balaguru	473797	64	M	7	6/18	6/18	17.3	17.3	N	A	P	A	IV	106/211	A	A	A	
123	Veerasamy	473890	62	M	5	6/18	6/18	20.6	18	N	A	A	A	IV	100/198	A	A	A	
124	Muthumanikam	473957	57	M	3	6/9	6/9	14.6	17.3	N	A	A	A	IV	88/150	A	A	A	
125	Jasmine	474053	51	F	2	6/9	6/9	14.6	17.3	N	A	A	A	IV	96/182	A	A	A	
126	Asokan	474222	64	M	5	6/12	6/12	17.3	20.6	N	A	A	A	IV	76/142	A	A	A	
127	Maasani	474350	60	M	5	6/12	6/12	18	20.6	N	A	A	A	IV	110/186	A	A	A	
128	Palanivel	474467	55	M	1	6/9	6/9	14.6	17.3	N	A	A	A	IV	112/198	A	A	A	
129	Ramanan	474508	40	M	0.1	6/6	6/6	14.6	17.3	N	A	A	A	IV	88/139	A	A	A	
130	Rajathi Ponnu	474686	55	F	7	6/9	6/12	16.5	17.3	N	A	P	A	IV	110/187	A	A	A	
131	Suryaprakash	474704	55	M	7	6/9	6/9	14.6	17.3	N	A	P	A	IV	100/222	A	A	A	

132	Ragavendran	474815	40	M	0.6	6/6	6/6	14.6	17.3	N	A	A	A	IV	96/160	A	A	A
133	Manikalingam	474950	53	M	4	6/12	6/12	13.8	15.1	N	A	A	A	IV	129/230	A	A	A
134	Muthupandi	475007	53	M	5	6/9	6/9	13.8	15.1	N	A	A	A	IV	88/146	A	A	A
135	Sundarapandian	505103	42	M	0.6	6/6	6/9	15.1	13.8	N	A	A	A	IV	108/187	A	A	A
136	Gnanavel	475222	61	M	6	6/6	6/6	25.1	23.1	N	P	P	P	IV	106/260	P	A	A
137	Chittupillai	475504	55	F	8	6/9	6/9	15.1	14.6	N	A	P	A	IV	98/160	A	A	A
138	Meenakshisundaram	475687	55	M	6	6/60	6/36	14.6	12.6	N	A	P	A	IV	110/118	A	A	A
139	Prasad	475740	42	M	1	6/18	6/18	14.6	12.6	N	A	A	A	IV	112/180	A	A	A
140	Pappu	475856	50	F	4	6/9	6/9	17.3	17.3	N	A	A	A	IV	86/160	A	A	A
141	Nellayappan	475920	50	M	6	6/9	6/12	14.7	17.3	N	A	A	A	IV	120/170	A	P	A
142	Periyapandi	476113	55	M	6	6/18	6/12	13.8	14.6	N	A	A	A	IV	78/140	A	A	A
143	Muniyandi	486720	42	M	1	6/6	6/6	12.6	15.1	N	A	A	A	IV	88/139	A	A	A
14	Muthuvelan	47672	66	M	7			17.	18	N	A	P	A	IV	97/189	A	A	A

4		8				6/12	6/24	3												
145	Sadayappan	476745	59	M	7	6/12	6/12	15.1	15.1	N	A	P	A	IV	96/180	A	A	A
146	Raja	476920	51	M	1	6/12	6/12	12.6	15.1	N	A	A	A	IV	100/176	A	A	A
147	Somasundaram	477003	40	M	0.2	6/36	6/24	14.6	17.3	N	A	A	A	IV	92/150	A	A	A
148	Sivakumar	488113	58	M	3	6/9	6/9	14.6	17.3	N	A	A	A	IV	129/200	A	A	A
149	Mani	477257	42	M	1	6/24	6/24	12.6	14.6	N	A	A	A	IV	88/164	A	A	A
150	Nafeesa Begum	477320	56	F	5	6/12	6/12	15.1	14.6	N	A	A	A	IV	96/150	A	A	A
151	Alagammal	477470	40	F	0.6	6/12	6/12	13.8	15.1	N	A	A	A	IV	111/180	A	A	A
152	Pandiammal	477555	43	F	1	6/9	6/9	14.6	17.3	N	A	A	A	IV	126/222	A	A	A
153	Karuppan	477674	45	M	2	6/9	6/9	14.6	17.3	N	A	A	A	IV	140/312	A	A	A
154	Subramaniam	477682	41	M	1	6/18	6/12	17.3	20.6	N	A	A	A	IV	88/150	A	A	A
155	Sengodan	477920	50	M	3	6/18	6/12	12.6	15.1	N	A	A	A	IV	76/136	A	A	A

156	Raaku	478133	49	M	2	6/9	6/9	17.3	20.6	N	A	A	A	IV	129/230	A	A	A
157	Sellathayi	478242	50	F	0.6	6/6	6/6	23.8	22.4	N	P	A	P	IV	225/307	P	A	A
158	Pechiammal	478302	53	F	6	6/6	6/6	20.6	22.4	N	A	A	A	IV	110/196	A	A	A
159	Pugalendi	478387	56	M	5	6/6	6/6	22.4	22.4	N	A	A	A	IV	76/150	A	A	A
160	Sellamma	478400	58	F	2	6/6	6/6	17.3	20.6	N	A	A	A	IV	160/320	A	A	A
161	Sundarajan	478540	41	M	2	6/6	6/6	20.6	17.3	N	A	A	A	IV	100/180	A	A	A
162	Velusamy	478678	56	M	7	6/9	6/9	17.3	14.6	N	A	A	A	IV	187/320	A	A	A
163	Azhagar	478916	50	M	4	6/9	6/9	15.1	17.3	N	A	A	A	IV	92/178	A	A	A
164	Shanmugam	478988	50	M	5	6/9	6/9	20.6	22.4	N	A	A	A	IV	89/162	A	A	A
165	Palkannan	479113	70	M	8	6/24	6/18	20.6	22.4	N	A	P	A	IV	108/187	A	A	A
166	Kannamma	479233	65	F	20	6/24	6/24	17.3	17.3	N	A	A	A	IV	110/186	A	A	A
167	Sivamani	479544	40	M	1.5	6/6	6/6	14.6	17.3	N	A	A	A	IV	68/142	A	A	A
168	Murugan	479623	50	M	3	6/12	6/9	14.6	16.5	N	P	P	P	IV	167/241	A	P	A
169	Muthuprasad	479709	44	M	1	6/6	6/6	14.6	14.6	N	A	A	A	IV	84/160	A	A	A
170	Thaipillai	479910	50	F	2			14.	17.	N	A	A	A	IV	88/158	A	A	A

0		7				6/18	6/18	6	3										
171	Veyila	480032	52	F	8	6/9	6/9	17.3	17.3	N	A	A	A	IV	104/186	A	A	A	
172	Ponnambalam	480257	48	M	8	6/18	6/60	22.4	23.8	N	A	P	A	IV	96/150	A	A	A	
173	Sivakami	480544	46	F	0.1	6/6	6/6	20.6	20.6	N	A	A	A	IV	135/270	A	A	A	
174	Santha	480566	52	F	8	6/6	6/6	17.3	20.6	N	A	P	A	IV	96/170	A	A	A	
175	Sargunam	480713	56	M	4	6/18	6/36	17.3	16.5	N	A	A	A	IV	120/198	A	A	A	
176	Chitrakkal	480905	45	F	7	6/6	6/6	20.6	22.4	N	A	A	A	IV	120/186	A	A	A	
177	Dhanalakshmi	481107	48	F	10	6/9	6/6	20.6	20.6	N	A	P	A	IV	118/190	A	A	A	
178	Chinnathangam	481323	46	F	0.6	6/9	6/9	20.1	23.8	N	A	P	A	IV	130/269	A	A	A	
179	Papathi	481400	40	F	4	6/6	6/6	17.3	17.3	N	A	A	A	IV	110/170	A	A	A	
180	Vellapandi	501522	55	M	10	6/18	6/18	31.8	25.1	N	P	P	P	IV	270/383	P	A	A	
181	Elango	481634	48	M	4	6/18	6/12	15.9	15.9	N	A	P	A	IV	96/142	A	A	A	
182	Sugapriya	481832	40	F	1	6/6	6/6	17.3	17.3	N	A	A	A	IV	85/199	A	A	A	
183	Kaleeswari	481903	45	F	1	6/1	6/1	17.3	17.3	N	A	A	A	IV	129/230	A	A	A	

							8	8										
184	Venugopal	482007	40	M	4	6/6	6/6	15.9	15.9	N	A	A	A	IV	167/226	A	A	A
185	Ellammal	482113	58	F	0.1	6/9	6/9	14.6	20.6	N	A	A	A	IV	170/300	A	A	A
186	Ponnusamy	482245	50	M	8	6/6	6/6	14.6	17.3	N	A	A	A	IV	96/140	A	A	A
187	Nagarathinam	482304	55	F	8	6/12	6/12	14.6	20.6	N	A	P	A	IV	110/186	A	A	A
188	Krishnan	482570	65	M	10	6/6	6/6	14.6	14.6	N	A	A	A	IV	102/193	A	A	A
189	Babu	482700	40	M	6	6/6	6/6	14.6	14.6	N	A	A	A	IV	160/220	A	A	A
190	Karuppi	482867	73	F	20	6/9	6/12	14.6	20.6	N	A	A	A	IV	116/186	A	A	A
191	Thangamma	482933	60	F	27	6/18	6/18	20.6	20.6	N	A	P	A	IV	114/190	A	A	A
192	Gandhimathi	482981	70	F	0.5	6/9	6/9	17.3	17.3	N	A	P	A	IV	100/202	A	A	A
193	Meenakshi	483027	44	F	2	6/6	6/6	20.6	22.4	N	A	N	A	IV	79/178	A	A	A
194	Christuraj	483167	51	M	5	6/12	6/12	17.3	24.4	N	A	P	A	IV	80/160	A	A	A
195	Ameer	503248	48	M	2	6/6	6/6	17.3	17.3	N	A	A	A	IV	100/168	A	A	A
196	Maragadham	483397	64	F	15	6/18	6/18	14.6	17.3	N	A	P	A	IV	140/220	A	A	A

197	Kuruvayi	483306	65	F	10	6/60	6/12	20.6	20.4	N	A	A	A	IV	110/175	A	A	A
198	Vellayan	483555	45	M	6	6/12	6/12	20.6	20.6	N	A	A	A	IV	120/186	A	A	A
199	Sinthamani	503612	52	F	3	6/6	6/6	20.6	20.6	N	A	P	A	IV	110/186	A	A	A
200	Muthu	483786	32	M	3	6/9	6/9	14.6	17.3	N	A	A	A	IV	76/110	A	A	A